MEDICAL LIBRARY
AUG 12 1937

The New England Journal of Medicine

Formerly The Boston Medical and Surgical Journal
Established 1828

Volume 217 JULY 29, 1937 Number 5

THE DIFFERENTIAL DIAGNOSIS OF JAUNDICE 161
Charles Gordon Heyd

POSTOPERATIVE PARATHYROID TETANY 165
Neil W. Swinton

A DIETARY STUDY IN RHEUMATIC FEVER 170
Sue E. Sadow; John P. Hubbard and *T. Duckett Jones*

MASSACHUSETTS MEDICAL SOCIETY 174
Proceedings of the Council

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL 187

EDITORIALS
Sir Squire Sprigge 193
Hollywood Surgery 194
Improved Care of Diabetics 194

DEPARTMENTS
Massachusetts Medical Society 195
Correspondence 195
Report of Meeting 196
Book Reviews 198

Owned and Published by the Massachusetts Medical Society

Published weekly in Boston at 8 Fenway. Domestic, $6 per annum, 25c per copy; Canada, $7.04; Foreign, $8.52.
Entered as second-class matter, November 16, 1932, at the postoffice at Boston, Mass., under the Act of March 3, 1879.
Copyright, 1937, by the Massachusetts Medical Society.

NEW HAMPSHIRE MEDICAL SOCIETY

OFFICERS FOR 1937-1938

PRESIDENT: SAMUEL T. LADD, Portsmouth.

VICE-PRESIDENT: CLARENCE O. COBURN, Manchester.

SECRETARY-TREASURER: CARLETON R. METCALF, Concord.

COUNCILORS: Clifton S. Abbott, Arthur W. Burnham, Frank M. Dinsmoor, Emery M. Fitch, Henry H. Amsden, Timothy F. Rock, Herbert L. Taylor, John A. Hunter, Richard E. Wilder, Charles E. Smith.

TRUSTEES: Alpha H. Harriman, 1938; Henry O. Smith, 1939; Frederic P. Lord, 1940.

HOUSE OF DELEGATES: Speaker, Richard W. Robinson; Vice-Speaker, W. J. Paul Dye.

DELEGATE A. M. A.: Deering G. Smith, 1937-1938.

ALTERNATE DELEGATE A. M. A.: Emery M. Fitch, 1937-1938.

NECROLOGIST: Henry H. Amsden.

STANDING COMMITTEES

ADVISORY COMMITTEE ON JURISPRUDENCE: Clifton S. Abbott, Fred E. Clow, Osmon H. Hubbard, Joseph J. Cobb, John F. Gile, Robert J. Graves, Herbert L. Taylor, Louis W. Flanders, Henry C. Sanders, Jr., Carleton R. Metcalf, Concord, Chairman.

AMENDMENTS TO CONSTITUTION AND BY-LAWS: Henry O. Smith, Fred E. Clow, Emery M. Fitch.

CONTROL OF CANCER: George C. Wilkins, Howard N. Kingsford, George F. Dwinell.

NEW ENGLAND MEDICAL COUNCIL: Samuel T. Ladd, President; Carleton R. Metcalf, Secretary-Treasurer; Robert H. Brooks, 1938; David W. Parker, 1939.

TUBERCULOSIS: Robert B. Kerr, Robert M. Deming, John D. Spring.

MEDICAL ECONOMICS: Frederic P. Lord, 1938; Timothy F. Rock, 1939; Leslie K. Sycamore, 1940.

MEDICAL EDUCATION AND HOSPITALS: John P. Bowler, James W. Jameson, Harris E. Powers.

MENTAL AND SOCIAL HYGIENE: Charles H. Dolloff, Benjamin W. Baker, Charles A. Weaver.

PUBLIC RELATIONS: Samuel T. Ladd, Carleton R. Metcalf, Robert J. Graves, John F. Holmes.

PUBLICATION: Carleton R. Metcalf, Henry H. Amsden, Warren H. Butterfield.

SCIENTIFIC WORK: Carleton R. Metcalf, Frederick P. Scribner, Richard W. Robinson.

SPECIAL COMMITTEES

ADVISORY COMMITTEE ON MEDICAL RELIEF: Robert J. Graves, John P. Bowler, Roland J. Joyce.

MATERNITY AND INFANCY: Robert O. Blood, Benjamin P. Burpee, Chester F. McGill.

CHILD HEALTH: Colin C. Stewart, Jr., Travis P. Burroughs, Franklin N. Rogers.

J. S. WATERMAN & SONS
SINCE 1832
FUNERAL SERVICE
Local and Suburban

Harvard Medical School
Courses for Graduates

Special courses for Commonwealth Fund Postgraduate Medical Fellows in Medicine, Pediatrics, Obstetrics and Office Surgery are offered for one month.

For further information apply to the Assistant Dean, Courses for Graduates, Harvard Medical School, Boston; Massachusetts

A New Product—Replacing Ferric Ammonium Citrate

FERROSATE

Tablets Ferrous Sulphate, exs. U.S.P. aa gr. IV

Clinical trials have shown that *3 tablets of Ferrosate daily are the equivalent of 90 grains of Ferric Ammonium Citrate in hemoglobin regenerating power,* and free from gastric disturbance sometimes accompanying the use of the citrate. Incorporated in a special stabilizing vehicle.

SUPPLIED IN BOTTLES OF 100
Also supplied in Elixir form

Kenmore Pharmacy, Inc.
500 COMMONWEALTH AVE., BOSTON

Support your advertisers.

The New England
Journal of Medicine

Copyright, 1937, by the Massachusetts Medical Society

VOLUME 217 JULY 29, 1937 NUMBER 5

THE DIFFERENTIAL DIAGNOSIS OF JAUNDICE

CHARLES GORDON HEYD, M.D.*

NEW YORK CITY

JAUNDICE as a symptom is as old as medicine itself. Galen gave noteworthy descriptions of clinical jaundice. Recently much has been written in regard to toxic hepatitis in arthritis, urticaria and eczema. In the industrial world jaundice as the direct result of toxic substances is arousing more clinical interest and assuming much medico-legal significance. The toxic hepatitis from arsphenamine is well known, as are the occasional cases following the ingestion of cinchophen. Of increasing importance is toxic hepatitis with jaundice following the use of fluids containing carbon tetrachloride in dry cleaning, and in cleaning motion-picture films. McNee's classification of jaundice remains the best and most workable division. There are three types: (1) obstructive; (2) toxic or infectious hepatic and (3) hemolytic.

In 557 patients whom I operated on for gall bladder disease, jaundice occurred in 16.3 per cent of the cases and was associated with pancreatitis in 3.7 per cent, yet only 4.6 per cent of the cases required exploration of the common duct, giving roughly 12 per cent with intrinsic disease of the gall bladder having jaundice at some time previous to or during hospitalization.

The differential interpretation of jaundice is based upon a consideration of the following diagnostic factors: (1) the history; (2) the routine examination of the stools over a period of at least five days, or a review of the findings from duodenal drainage over a similar period of time; (3) physical examination—the presence or absence of Murphy's inspiratory spasm; (4) x-ray examination; (5) blood chemistry—embracing the van den Bergh test, the icteric index, urea nitrogen, cholesterol and cholesterol ester determinations, and the galactose test of Bauer.

Our conception of jaundice has undergone many changes and recently a fundamental revision. In 1847 Virchow upon finding hematoidin crystals in connection with fluid collections of blood postulated the development of jaundice from causes that arose within the liver, and in addition, a jaundice that arose from pathologic processes outside of the liver. This view was held more or less intermittently thereafter, although it was largely invalidated by the celebrated experiments of Minkowski and Naunyn, carried out on geese, the livers of which had been removed and in which despite the severest degree of arsenhydrogen poisoning no jaundice occurred. In the light of these experiments it was asserted: "Without the liver, no formation of bile; without the liver, no jaundice." Thus the only type of jaundice that could be recognized was that of hepatogenous origin, it being considered that true hematogenous icterus was not possible.

The blood content of bilirubin may be raised to 1 in 40,000 before macroscopic tinging or tissue jaundice occurs. There is thus a blood icterus which ranges from the normal blood bilirubin content up to a bilirubinemia of 1 in 40,000 before clinical jaundice is apparent. This bilirubin range is termed latent jaundice—an icterus of the blood serum, detected only by chemical tests. It is obvious that a blood icterus always anticipates a clinical icterus. This chemical blood condition is analogous to that which is present in diabetes with blood sugar and in nephritis with blood urea. In a chemical sense jaundice is essentially a disorder of bilirubin metabolism, and always manifests itself by an accumulation of bilirubin above the normal physiologic blood content.

Jaundice has been defined as a visible coloration of the skin, mucous membranes and sclerae, due to the deposition of bile pigments. In this form, however, we recognize jaundice as a clinical or tissue jaundice, which gives a very imperfect picture of the underlying chemical mechanism. Jaundice is an expression of the sum total of the retention and accumulation of bilirubin in the blood stream. There is at all times circulating in the

Abstract of lecture delivered at the annual meeting of the New Hampshire Medical Society, Manchester, New Hampshire, Wednesday, May 19, 1937.

*Professor of surgery, New York Post-Graduate Medical School, Columbia University, New York City.

blood stream a normal content of bilirubin. This amount varies from 1 part of bilirubin in 400,000 to 600,000 parts of blood serum and is termed "physiologic bilirubin." As previously stated, the blood stream may contain a content of bilirubin equal to 1 in 40,000 before the skin becomes tinged or clinical jaundice appears.

The intensity of the jaundice can be estimated by the determination of the icterus index; this is the measure of intensity of the color of the blood serum as compared with a standard 1:10,000 solution of potassium bichromate. Under normal circumstances the normal icteric index varies from 3 to 6; the clinical evidences of jaundice are usually obtained when the icteric index rises to 20 or more. The interval between 6 and 20 is known as a period of latent jaundice, when there is more than the normal amount of bilirubin in the blood stream, yet not enough to give an icteric tinge to the skin and sclerae.

There is another aspect of jaundice which must be canvassed. Is the jaundice due to a retention of bilirubin with or without the bile acids? Retention with bile acids signifies that the causal factor is in some way or another obstructive, and due to the reabsorption of a completely formed bile. A jaundice that is due to the retention of bilirubin per se and without bile acids is in no way connected with an obstructive phenomenon, and is referred to as a "dissociated jaundice." This is the type that occurs during the absorption of large hemorrhagic exudates, and also in certain types of familial jaundice.

Van den Bergh assumed that icterus was entirely due to an increase of bilirubin in the serum, and upon utilizing the color reactions given by bilirubin with the diazo reagent of Ehrlich, he was able to make the original observation that in some cases it was necessary to bring the bilirubin into alcoholic solution before a color reaction could be obtained, while in others the adding of the icteric serum directly to the diazo reagent brought about a color reaction. He therefore made the very important distinction that in the icteric group there were two distinct kinds of serums: first, serum which gave a color reaction at once or immediately upon the addition of diazo reagent directly to the serum, the so-called "prompt or immediate reaction;" secondly, serum which gave no color reaction, or only after a long delay, on the addition of the diazo reagent directly to the serum, the so-called "delayed reaction." Van den Bergh supposed that a prompt direct reaction was obtained only from a bilirubin that had passed through the polygonal liver cells and had subsequently been absorbed because of obstruction. On the other hand, if the serums gave a delayed reaction he assumed that the bilirubin had been formed independently of the liver cells, and in addition had not passed through them.

If this thesis be correct, then there are two kinds of bilirubin that may cause jaundice. That this contention is supported by clinical facts is evidenced by the following: (1) bile from the gall bladder always gives a typical prompt direct reaction—this bile has obviously passed through the liver; (2) bilirubin obtained from the serous fluids in hemorrhagic effusions in the chest, abdomen or knee joints, may give the delayed but generally gives no reaction. Such a bile pigment has been elaborated independently of the polygonal cells of the liver.

It is the cells of the reticuloendothelial cell system, and particularly Kupffer's cells in the liver and the reticular cells of the spleen, that are concerned with the breaking down of hemoglobin into bilirubin. Bile pigment, hemosiderin and fragments of red corpuscles have repeatedly been seen in both these types of cell, but particularly in Kupffer's cells. It would seem, then, that reticular tissue without and within the liver (Kupffer's cells), and not the parenchymal liver cells, plays a very prominent part in the formation of bile pigment. On the other hand, not the slightest proof has been brought forward to substantiate the hepatocellular formation of bile pigment. Jaundice may, then, theoretically arise from the obstruction of the outlet of the bile capillaries after the changed bilirubin has passed through the polygonal cells of the liver, or from the fact that the bilirubin has not passed through the polygonal cells but is absorbed by the vascular capillaries or lymphatics of the liver into the general circulation. In the former case, there would be elimination of an altered bilirubin with bile salts and subsequent absorption from obstruction; in the latter, there would be the absorption of an unaltered bile pigment without bile salts.

It is this conception of jaundice that explains the two reactions of van den Bergh's test. It seems possible therefore to predicate inferentially two different kinds of bilirubin. While these have not been experimentally or clinically demonstrated, the behavior of bilirubin in varied conditions of jaundice allows this assumption. It would seem that a normal content of bilirubin is circulating at all times in the body. A portion of this, which we may call bilirubin A, is taken out by the activity of the liver cells and eliminated in the bile. It is possibly of no use to the organisms and represents a waste product in the process of excretion, for by its concentration or by some slight change in its chemical character, it gives the positive direct reaction of van den Bergh. The remaining moiety

of bilirubin is evidently a useful product conserved by the organism, and so is not withdrawn from the blood stream by the liver cells but continues circulating; it is dissimilar either in concentration or in slight chemical variation to the other, and ordinarily gives the indirect reaction of van den Bergh. Apparently this type of bilirubin reaches a large measure of concentration in pathologic hemolysis and gives the distinct bilirubinemia of hemolytic jaundice. The amount of bilirubin in hemolytic jaundice available for action upon by the liver cells is greatly in excess of their capacity, and there may be a direct injury to the liver cells by reason of the overcharged bilirubin content. The presence of calculi in hemolytic jaundice in non-inflammatory gall bladders and ducts would suggest that this element of pleiochromia was a factor in their production. The removal of the spleen would obviously limit the degree of pathologic hemolysis, and the liver would then be able to excrete its normal quantity of bilirubin.

The liver has a threshold for the elimination of bilirubin just as has the kidney. In some cases it allows the elimination of bilirubin in the bile much more readily than in others, and in hemolytic jaundice the threshold is exceedingly high with retention and accumulation of bilirubin in the blood stream. The bilirubin that occurs in frank obstructive jaundice or in disease of the liver cells has a renal threshold of 1:40,000, whereas the bilirubin that occurs after marked hemolysis, and meets its best example in hemolytic jaundice, has a much higher threshold of renal elimination and does not appear in the urine as a bilirubinuria. In fact, it may be doubted if in hemolytic jaundice, without some added complication, bilirubin ever passes through the kidney at all.

A differential diagnosis of jaundice will embrace an analysis of the history and examinations of the gastrointestinal tract, blood and urine, together with a physical examination of the patient and such additional information as may be given by x-ray studies. Statistical analysis of a large series of cases exhibiting jaundice indicates that according to frequency jaundice may be classified in the following order: as associated with (1) gallstones and infection of the extrahepatic bile ducts; (2) catarrhal jaundice—hepatic degeneration; (3) cancer of the liver; (4) cirrhosis of the liver; (5) cancer of the bile ducts or gall bladder; (6) cancer of the pancreas; (7) cancer of the stomach.

Of outstanding importance is whether the onset of the icterus is associated with pain. This is of such significance that most clinicians for ease of diagnostic interpretation speak of "painless jaundice" and "jaundice associated with pain." The pain may be either of two types:

(1) Colicky. This pain comes on suddenly, has a rapid rise to a maximum intensity, is followed by a short free interval from pain, and is so severe as to require anodynes. It is pre-eminently the type associated with calculus or infectious obstruction of the cystic, hepatic or common duct.

(2) Distention. This is a dull, heavy, "stretching pain." It is continuous, has none of the colicky or paroxysmal features of the former type, and is associated with the stretching of the capsule of Glisson from hepatic degeneration, with malignancy or metastatic deposits in the liver. A distended gall bladder from carcinoma of the head of the pancreas gives the same general type of pain.

The onset of jaundice with colicky pain is abrupt, and the icterus appears within twenty-four hours. Jaundice that appears painlessly is insidious and continuous, and ordinarily some neighbor or member of the family informs the patient that he has a yellow color. To recapitulate, the development of jaundice after a sudden attack of colicky pain presupposes disease of the external biliary apparatus, in which calculus and infection are the etiologic factors. A slow, creeping jaundice without initiatory pain which continues to rise in intensity, with the development of an aching type of pain some time after the onset of jaundice, is indicative of either a neoplastic compression of the common duct or the development of some intrinsic pathology of the liver.

In all infectious conditions of the gall bladder there is a concomitant infection of the common duct and hepatic ducts, and mechanical obstruction, be it caused by calculous or infectious products, is as a rule intermittent and rarely complete for long periods of time. In a malignancy of the head of the pancreas or of the common duct, the occlusive process is complete and permanent. In hepatic degenerations, which vary from catarrhal jaundice to acute yellow atrophy, there is no change in the lumen of the extrahepatic ducts sufficient to cause obstruction, and therefore an investigation as to the presence or absence of bile in the gastrointestinal tract is of great diagnostic importance.

Either of two methods of investigation may be carried out: daily examination of the stool for the presence or absence of bile, or duodenal drainage for three successive days. A complete absence of bile from the stools or in the duodenal drainage in patients with continuous jaundice suggests a malignant obstruction of the external biliary duct system; its intermittent presence suggests that from time to time bile is being delivered into the intestinal tract, and therefore points to inflammatory disease, with or without calculi, of the ex-

ternal biliary duct system or intrinsic disease of the liver.

Certain data may be obtained from blood chemistry. Of outstanding importance is the periodic determination of the icteric index. Since the liver is concerned with the excretion of bile pigment (bilirubin), carbohydrate metabolism and the analysis and synthesis of protein, it follows that examination of the chemistry of the blood may be fruitful in suggesting diagnostic differentiation. The icteric index, as already stated, determines the intensity of jaundice; clinical evidences of this disease are usually present when it reaches 17. The index represents a quantitative estimation of icterus, and gives no information as to whether the jaundice is obstructive, toxic or hemolytic. However, in obstructive jaundice the icteric index rises rapidly and maintains itself in a more or less uniform elevation. In malignancy of the head of the pancreas it is usually from 150 to 250, while the elevation in hepatic degeneration is continuous and has a tendency to rise above 300. The icteric index may be combined with the van den Bergh, but the determination as to whether the reaction is direct or indirect is not of much diagnostic importance.

Bauer has suggested the galactose tolerance test. When galactose is ingested the liver converts it into glycogen and ultimately into dextrose. The kidney has no threshold for galactose, and so long as it is circulating in the blood it will be excreted in the urine. If 40 gm. of galactose are taken by the patient on an empty stomach, the liver readily metabolizes it so that less than 1 gm. and rarely more than 3 gm. will appear in the urine in a five-hour period. If liver degeneration is present galactose appears in larger amounts in the blood stream, and the finding of more than 3 gm. in the urine in a five-hour period suggests liver degeneration. In obstructive jaundice there is no inability of the liver to metabolize galactose. In late cases of liver disease with edema and hydrohepatosis, the galactose test will indicate liver disability.

Where precise laboratory facilities are available the chemical determination of cholesterol and cholesterol esters is of value. The normal blood contains about 200 mg. of cholesterol per cent. Under normal liver physiology the cholesterol, which is an alcohol, is united with palmitic, stearic and oleic acids to form cholesterol esters. There is an enzyme substance—esterase—in the liver which in turn breaks down cholesterol esters into cholesterol and fatty acids. As only cholesterol is excreted in the bile it follows that with complete biliary obstruction it accumulates in the blood, and the determinations rise to 300 or 400 mg. per cent. In hepatic degeneration the esterase activity is inhibited and the cholesterol esters drop or may disappear. In the presence of jaundice the persistent absence of cholesterol esters from the blood is grave prognostic evidence pointing to serious damage to the liver parenchyma.

The presence of petechiae or the development of purpuric spots along the lines of a lightly applied tourniquet is significant of the hemorrhagic tendency in jaundice. Pruritus may be present. Contrary to opinions expressed in the literature, our own experience has been that chronic jaundice due to neoplasm of the liver is not associated with marked pruritus. The most marked cases of pruritus have been those associated with the obstruction due to calculus in the extrahepatic duct system.

The presence or absence of ascites suggesting late portal cirrhosis or metastatic malignancy is readily determined. In the absence of ascites the liver edge may be palpated and significant information may be obtained. Not infrequently, bosselated areas of nodular deformities of the liver may be felt; this is significant of metastatic carcinoma of the liver. In approximately 80 per cent of the malignancies of the head of the pancreas, a pear-shaped tumor may be felt in the right upper quadrant. This tumor—a distended gall bladder—increases in size with the jaundice and is the basis of Courvoisier's law: "In cases of chronic jaundice due to blocking of the common duct, a contraction of the gall bladder signifies that the obstruction is due to stone: a dilatation of the gall bladder that the obstruction is due to causes other than stone."

Jaundice plus pain, with bile in the stool or duodenal drainage, denotes calculous or infectious obstruction of the external biliary system. Jaundice with neither pain nor bile in the stool or duodenal contents indicates carcinoma of the external biliary system. Jaundice without pain, with bile in the stools or duodenal contents, indicates carcinoma of the external biliary system or intrinsic pathology of the liver. Hemolytic jaundice, familial jaundice and the jaundice of the anemias, are all termed "dissociated jaundice" in that there is an excess of bilirubin in the blood but no retention of bile salts or bile acids, and the excretory function of the liver for the delivery of bile into the intestinal tract is normal. Gallstones, however, may be found at laparotomy in patients with hemolytic jaundice because there is "bile retention" in the intrahepatic bile ducts due to the excess of bile pigments, and to the inability of the liver to excrete this excess. Bile thrombi occur in the intrahepatic bile canaliculi, and there is a deposition of bile pigments in the gall bladder with the production of pleiochromic calculi.

POSTOPERATIVE PARATHYROID TETANY

NEIL W. SWINTON, M.D.*

BOSTON

POSTOPERATIVE parathyroid tetany has been an infrequent but distressing complication of thyroid surgery since the advent of subtotal thyroidectomy for the relief of hyperthyroidism. Parathormone, the active principle of the parathyroid glands, has been satisfactory in the control of the acute phases of the disease, but it has become apparent that this agent has not been entirely satisfactory in the control of chronic cases.[1, 2] New interest has been created by the preliminary report of Holtz[3] and his co-workers in 1933 on the use of dihydrotachysterol, or AT-10, in the treatment of this condition.

From 1915 to 1937, there were 15,552 thyroid operations performed on 13,000 patients at the Lahey Clinic; 26 cases of both transient and permanent parathyroid tetany followed these operations—an incidence of 0.2 per cent. I propose to report this group, to review the methods by which postoperative incidence may be kept at a minimum, and to discuss the symptomatology, diagnosis and present attitude toward the treatment of the disease.

McCullagh,[4] in 1934, reported an incidence of 1.3 per cent in a series of 11,508 patients operated upon for thyroid disease. Boothby,[5] in 1931, reported 88 cases in a series of 13,300 thyroid operations, an incidence of 1.5 per cent. In our series it is of interest that no cases were found in males, although males constituted one quarter of all cases of operated thyroid disease in the clinic. The rarity of this disease in males has been noted by other observers.[4, 5] Because of technical difficulties, it seems obvious that postoperative parathyroid tetany would be commoner following recurrent operations on the thyroid gland, and 10 of the 26 patients in this series had had previous thyroidectomy.

Postoperative parathyroid tetany results from injuring or removing the parathyroid glands. Its incidence can be kept to a minimum if the parathyroid glands are recognized when encountered at the time of operation and their injury or removal is avoided. It has always been a fundamental principle in this clinic to obtain an adequate exposure of the thyroid gland itself and of its adjacent structures at the time of operation. A low-collar incision is made, and the skin flap, including the platysma muscle, is elevated to a high level above the notch of the thyroid cartilage. The sternomastoid muscles are separated from the deep cervical fascia, and the prethyroid muscles, after having been split in the midline, are divided transversely at a high level (Fig. 1). This procedure allows a complete exposure of the entire anterior surface of the gland (Fig. 2). By next dividing the middle and inferior thyroid veins and retracting the sternomastoid muscles and the carotid sheath with its contents laterally and the gland medially, an adequate exposure of the lateral and posterior surface of the gland is obtained (Fig. 3).

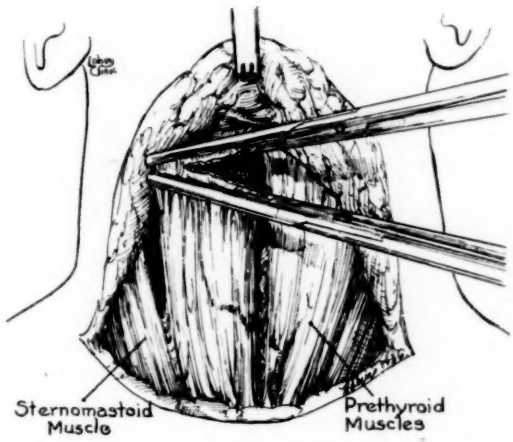


FIG. 1. The elevation of the skin flap and platysma muscles to a level above the notch of the thyroid cartilage. The prethyroid muscles are next divided at a high level.

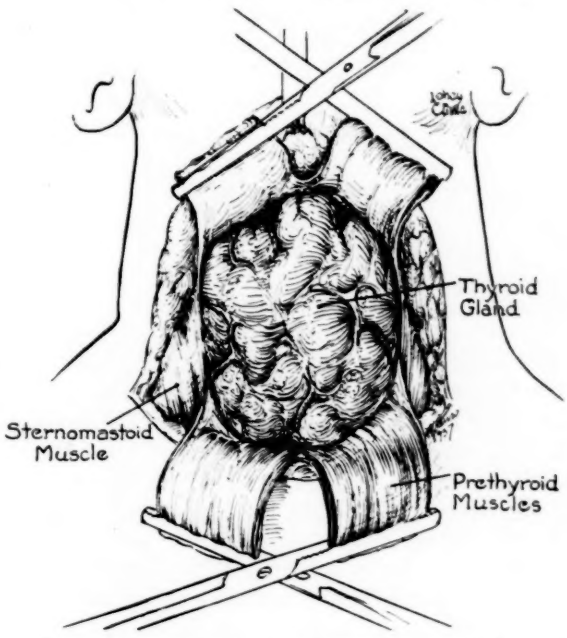


FIG. 2. The exposure of the anterior surface of the thyroid gland.

*Surgeon, Lahey Clinic.

In this way the parathyroid bodies can usually be identified and guarded.

The parathyroid glands are usually four in number consisting of two superior and two inferior

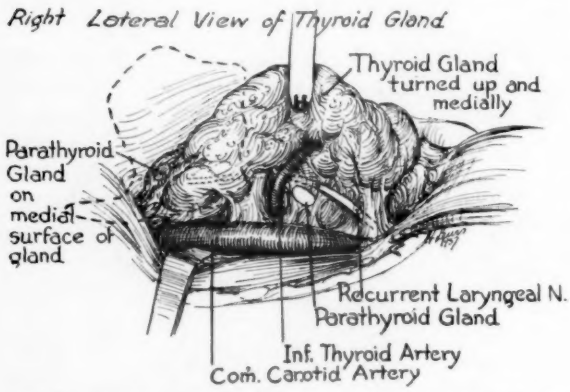

Right Lateral View of Thyroid Gland
Thyroid Gland turned up and medially
Parathyroid Gland on medial surface of gland
Recurrent Laryngeal N.
Parathyroid Gland
Inf. Thyroid Artery
Com. Carotid Artery

FIG. 3. Lateral view of the thyroid gland: the superior thyroid vessels have been ligated, the thyroid gland has been retracted medially up over the trachea, and the sternomastoid muscles and great vessels have been retracted laterally. The inferior parathyroid gland is seen in its usual position near the junction of the recurrent laryngeal nerve and the inferior thyroid artery. The superior parathyroid gland is seen in a very common location, just behind the superior pole of the thyroid.

glands on either side of the thyroid. They are usually mahogany brown, but this color varies according to age, being slightly darker in older people. The glands are usually slightly flattened and oval-shaped, and vary from 3 to 6 mm. in width and from 4 to 9 mm. in length. Owing particularly to the interest exhibited in disease associated with adenomas of the parathyroid gland, many observers have reported on the location, blood and nerve supply of these bodies.[6, 7, 8] In our series of goiter operations, over 200 parathyroid glands were found on the surgically removed specimens. These glands were recovered immediately, and after sections had been taken for the pathologists and their identity had been proved, they were transplanted back into the sternomastoid muscle, as suggested by Lahey.[16]

The location of the parathyroid glands varies greatly. They have been found on the anterior surface of the thyroid gland in many instances. We have recovered them imbedded in the substance of the thyroid gland, and the removal of an adenoma of a parathyroid from the mediastinum[7] has been reported. Herrman[9] has recently described the position of the inferior parathyroid bodies as lying at the end of a strand about 5 mm. in length formed by a branch of the inferior thyroid artery, which has been joined with a branch of the recurrent laryngeal nerve, very near the point where the inferior thyroid artery crosses over the recurrent laryngeal nerve. In Herrman's series, 34 of the 44 cases studied showed the inferior parathyroid bodies to lie in this position, and in our experience they have usually been found in this vicinity.

Herrman[9] describes the superior parathyroid bodies as lying at a point behind the superior pole of the thyroid at the end of a strand formed by a branch of the superior thyroid artery, which frequently has an anastomosing branch from the inferior thyroid artery, and a branch of the superior laryngeal nerve. We have found that these glands can usually be found lying along the trachea, behind the superior pole of the thyroid gland, at about a level of the junction of the upper and middle thirds of the thyroid (Fig. 4).

The amount of parathyroid tissue that can be removed without the production of clinical tetany is unknown. The number and location of such bod-

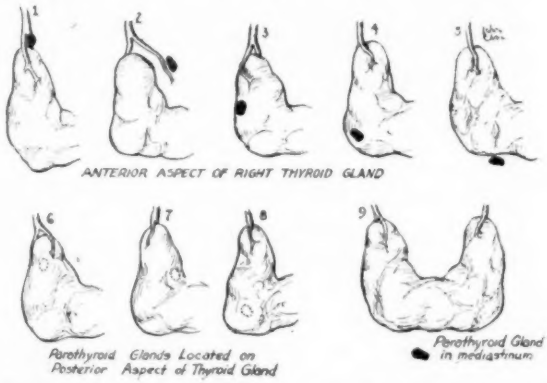


FIG. 4. Typical locations of the parathyroid glands. Drawings 1 to 6 illustrate common locations on the anterior surface of the thyroid gland. Drawings 6 to 9 illustrate typical locations on the posterior surface of the thyroid gland. In Drawing 9 a gland is seen in the superior mediastinum.

ies around the thyroid gland is so variable that in thyroid surgery one must be constantly alert to prevent removing or injuring them. On several occasions at this clinic two parathyroid glands have inadvertently been removed at the time of a subtotal thyroidectomy without the production of tetany. A method to control hemorrhage from a remaining remnant of thyroid tissue which has been followed at the clinic for a number of years is to ligate the main trunk of the inferior thyroid artery outside the recurrent nerve and the substance of the thyroid gland itself. It is believed that this procedure is safer to control bleeding from the small remnants of tissue than to thrust sharp-pointed hemostats blindly down along the side of the trachea into the gland substance, where the recurrent nerve can so easily be injured. It has not infrequently been found necessary to ligate the main trunks of both inferior thyroid arteries even after both superior thyroid vessels have been ligated. Postoperative parathyroid tetany has never developed following this procedure. However, as the remaining number of parathyroid bodies and their exact location are never known, the removal of or the interference with the blood supply to any parathyroid tissue is regarded as dangerous and to be avoided when possible.

The hormone of the parathyroid glands controls the calcium and phosphorus metabolism of the

body. Its loss produces the clinical condition known as tetany, characterized by hyperirritability of the nerve cells. This is apparently due to a decrease in available calcium, and is manifested by paresthesia and muscle spasm, which may be constant or intermittent, local or generalized. In the long-standing cases, trophic changes develop, and are usually seen first in the lens of the eye, the nails and the teeth.

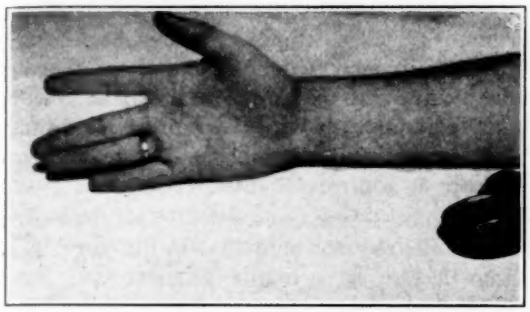


FIG. 5. The normal hand is seen; a blood pressure cuff is being applied to the upper arm.

The time of onset of symptoms of tetany is variable. The majority of cases in this series developed clinical tetany between 2 and 7 days following operation. One patient, however, did not develop any symptoms suggestive of tetany until 3 weeks had elapsed. The symptom usually noted first was numbness or tingling of the lips, nose or hands. One patient, on the third day after the removal of a huge bilateral intrathoracic goiter, complained bitterly for several hours of a numbness and tingling of her nose. It was not until she had developed generalized convulsions that the presence of parathyroid tetany was appreciated. An unnatural flexion of the hands, wrists and elbows in a postoperative thyroid patient should always lead one to suspect the presence of parathyroid tetany; if one straightens out the hands and arms they will be seen to slowly resume their previous flexed positions. Typical examples of these characteristic positions may be seen in the accompanying photographs (Figs. 5-8), which demonstrate the use of Trousseau's sign in the detection of postoperative tetany and show a typical type of contraction of the hands. Fine fibrillary tremors of the face and the muscles of the extremities are seen as the disease progresses. A flushed, apprehensive facial appearance with a typical circumoral pallor is often present. Vision and speech may be blurred. As the laryngeal muscles become involved, there may be a marked respiratory stridor and the presence of a recurrent contracture of the abdominal muscles. Late in the disease the entire body becomes hyperextended in a generalized convulsion. Coma and death may result or the attack may subside to recur later. We have never had a case of tetany of this severity, nor a death from tetany in this clinic.

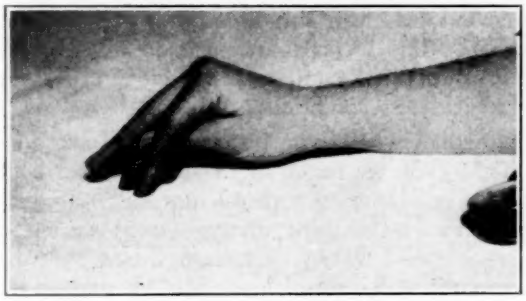


FIG. 6. Pressure in the blood pressure cuff has been raised to 200 mm. of mercury, and a beginning contraction of the right hand is seen.

FIG. 7. Typical "obstetric hand" type of contraction seen so frequently in postoperative parathyroid tetany.

Chvostek's and Trousseau's signs are nearly always present and are easily elicited. The phenomena described by Pool and Schlesinger may aid in the detection of the disease. The most constant sign is the demonstration of nerve hyperexcitability by the use of galvanic stimulation (Erb's phenomenon). It is seldom necessary to resort to this sign yet it may occasionally be useful in borderline cases. The demonstration of a lowered blood-calcium content and an elevated blood-phosphorus content with any of the above clinical signs establishes a diagnosis of postoperative parathyroid tetany.

The severity of the acute attack does not seem to have any accurate relation to its duration. One of our patients developed a very severe parathyroid tetany on her third postoperative day; she had a generalized convulsion, which was accompanied by coma, a blood calcium determination of 5.2 mg. per cent and a blood phosphorus of 5.8 mg.

per cent. Her acute symptoms were immediately relieved by the use of parathormone and calcium. Six weeks later all medication was permanently discontinued. She has maintained a normal blood calcium and phosphorus ratio and has been perfectly well for the past 2½ years. Fifteen of our 26 cases have required some form of calcium medi-

FIG. 8. The pressure in the blood pressure cuff after having been maintained for 5 minutes has been released. Note that the opposite hand also shows a tendency to go into spasm.

cation for a period of over 1 year. Six cases, which were followed for over a period of a year since the discontinuation of any form of therapy, have been found to have normal blood-calcium and phosphorus determinations and no clinical evidence of parathyroid tetany. These were undoubtedly cases of transient tetany. Five of our cases have been of too recent development to group accurately as yet.

The symptoms of chronic or latent tetany may be so mild as to be overlooked for a long time. Intermittent contractions, stiffness of the hands, numbness of the face and paresthesia may be the only clinical manifestations of the disease. Two of our patients with long-standing tetany have noted an unusual relation of the symptoms to pregnancy and menstruation.[10] Dulling of the mental processes has been observed in other cases, although we have not noted this characteristic in the patients in our series. We have had 2 patients with long-standing tetany, however, who we now feel were not properly treated during the early years of the disease, and who developed marked trophic changes of the nails and hair, and cataracts, for which operation was necessary.[11]

The therapeutic goal is to relieve clinical manifestations and return the calcium and phosphorus contents of the blood serum to their normal levels. This treatment is concerned with three substances: the active principle of the parathyroid gland (parathormone), calcium and vitamin D substances. The nerve hyperirritability is due to a lack of available calcium, and treatment of parathyroid tetany therefore centers around this substance. Albright[12] has shown that the primary effect of parathormone is to lower the blood-phosphorus content by causing an increased excretion of this element in the urine, and that the effect on the calcium level is secondary. It has been shown that vitamin D preparations aid the absorption of calcium and thus help to control the disease.[5, 13, 14]

In acute and severe cases, calcium must be given in large and frequent amounts; in our experience, calcium lactate is a highly effective and easily available form. In acute cases we have given 1 to 2 heaping teaspoonfuls (120 to 240 gr.) of calcium lactate every hour until symptoms have been controlled. If necessary, calcium gluconate or calcium chloride (10 per cent solution) may be given intravenously. The treatment of parathyroid tetany should always be controlled by frequent blood-calcium and phosphorus determinations. In severe, acute cases, there may not be time or facilities to obtain these determinations quickly, and therapy should be instituted as early as possible. Calcium lactate given by mouth is readily absorbed. If the attack is severe and if generalized convulsions and coma are present, however, it is best to give calcium intravenously immediately, in the form of calcium chloride or calcium gluconate solution. The intravenous injection of either solution results in a rapid and marked improvement in the patient's condition.

Immediately after the introduction of parathormone in 1926, we began to employ it in our acute cases. In combination with calcium, small amounts were given at frequent intervals until the attack had subsided. Lately, we have found that calcium alone will control acute attacks, and in our more recent cases parathormone has been unnecessary. Five of our early cases of chronic tetany were treated with parathormone and calcium. Two of these, in spite of this treatment, developed the severe trophic changes previously mentioned. Certain cases apparently develop a refractivity to parathormone,[1-2] and this substance has not been entirely satisfactory in the control of long-standing cases. A danger that may result from the long-continued use of parathormone if it is not accompanied by an adequate calcium intake is bone demineralization.

During recent years our patients with chronic tetany have been treated with large amounts of calcium in the form of calcium lactate by mouth, a low phosphorus, hence a low calcium diet and small amounts of viosterol. In most of the patients the disease has been fairly well controlled,

yet many at the time of their menses, and occasionally at other times, have noted some paresthesias and tingling sensations. A recent study of the blood chemistry in such patients has shown that their blood-calcium and phosphorus contents were not quite normal.

In 1933, Holtz, of Berlin, first demonstrated the substance, dihydrotachysterol. This is a fraction which he found in breaking up vitamin D; it has a calcium-elevating effect and is less toxic than vitamin D itself and, when mixed with an oily solvent, is called AT-10. It was first used in this country, so far as we know, by Arnold and Blum in 1935. They have recently reported on its use in 26 cases.[15]

Six of our cases of chronic postoperative parathyroid tetany were started on AT-10 in October, 1936.* To date, with the possible exception of 1 case, these patients have felt better, and their blood-calcium and phosphorus determinations have more nearly approached normal than under any other form of treatment. The material has the great advantage of being taken by mouth. It accomplishes the elevation of blood calcium by influencing the patient's ability to utilize administered calcium rather than decalcifying bone. Quite accurate control of the level of blood calcium is possible and the psychic effect of oral administration, together with its ability to control symptoms, has a very distinct advantage over hypodermically administered remedies in that these patients are less liable to think of themselves as abnormal individuals. Our cases which have been under this treatment will be reported later in detail.

SUMMARY

1. An incidence of 0.2 per cent of postoperative parathyroid tetany is reported in a series of 13,000 patients operated on for thyroid disease.
2. The incidence of the disease is low if an adequate exposure of the thyroid gland and adjacent structures is obtained at the time of subtotal thyroidectomy, if the parathyroid glands are identified and if their removal or injury is avoided.
3. The symptomatology presented in this series of 26 cases is discussed.
4. Some experiences with the use of calcium and parathormone are presented.
5. Mention of the new vitamin D substance, AT-10, is made together with the suggestion that at present it seems to be the most satisfactory measure with which to maintain the normal calcium and phosphorus levels and to control tetany.

*We are greatly indebted to the Winthrop Chemical Company for supplying us with AT-10 for experimental purposes.

REFERENCES

1. Freyberg, R. H.; Grant, R. L., and Robb, M. A.: Hypoparathyroidism; the treatment of chronic cases. J. A. M. A. **107**:1769 (Nov. 28) 1936.
2. Collip, J. B.: Recent studies on anti-hormones. Ann. Int. Med. **9**:150 (Aug.) 1935.
3. Holtz, F.; Gissel, H., and Rossmann, E.: with clinical reports by Kramer, F.; Messmann, A.; Quadfasel, F., and Roggenbou, Chr: Experimentelle und klinische Studien zur Behandlung der postoperativen Tetanie mit A. T. 10. Deutsche Ztschr. f. Chir. **242**:521 (Mar.) 1934.
4. McCullagh, E. P.: Remarks on the management of parathyroid tetany. M. Clin. North America **17**:951 (Jan.) 1934.
5. Boothby, W. M.; Haines, S. F., and Pemberton, J. de J.: Postoperative parathyroid insufficiency. Am. J. M. Sc. **181**:81 (Jan.) 1931.
6. Morgan, J. R. E.: The parathyroid glands; study of normal gland. Arch. Path. **21**:10 (Jan.) 1936.
7. Lahey, F. H., and Haggart, G. E.: Hyperparathyroidism; clinical diagnosis and operative technique of parathyroidectomy. Surg., Gynec. Obst. **60**:1033 (June) 1935.
8. Hinton, J. W.: Location and preservation of parathyroid glands. Am. J. Surg. **30**:400 (Dec.) 1935.
9. Herrman, C. S.: Parathyroids: a new anatomic concept. J. A. M. A. **106**:1357 (April 18) 1936.
10. Swinton, N. W., and Claiborne, T. S.: Postoperative parathyroid tetany. S. Clin. North America **16**:1601 (Dec.) 1936.
11. Cattell, R. B.: Eye complications in exophthalmic goitre. Ann. Surg. **100**:284 (Aug.) 1934.
12. Albright, F.; Bauer, W.; Ropes, M., and Aub, J. C.: Studies of calcium and phosphorus metabolism; the effect of the parathyroid hormone. J. Clin. Investigation **7**:139 (April) 1929.
13. Boothby, W. M.: Treatment of postoperative parathyroid insufficiency. Proc. Staff Meet., Mayo Clin. **10**:87 (Feb. 6) 1935.
14. Stacey, R. S.: Treatment of low-calcium tetany with calciferol. Lancet **2**:656 (Sept. 21) 1935.
15. Arnold, C. H., and Blum, H.: The control of hypoparathyroidism. Tr. Am. A. Study Goitre. Pp. 79-88, 1936.
16. Lahey, F. H.: Transplantation of parathyroids in partial thyroidectomy. Surg., Gynec. Obst. **42**:508 (April) 1926.

FUNDS FOR THE AMERICAN SOCIAL HYGIENE ASSOCIATION

Demands upon the American Social Hygiene Association for its services have increased so sharply during the past six months that this national voluntary organization against syphilis and gonorrhea must raise at least $300,000 annually to do its work, said Dr. William F. Snow, general director of the association, in a statement made public recently.

Activities resulting from "Social Hygiene Day," promoted by the association and observed nationally in February; from Surgeon General Parran's attack on venereal diseases; and from the wide publicity in newspapers and magazines and by radio, all have stimulated an unprecedented volume of requests at association headquarters, 50 West 50th Street, New York City, according to Dr. Snow.

"Groups and individuals throughout the country, aroused at last to the dangers from syphilis and gonorrhea, have been pouring in requests for programs of community action, consultant service, surveys, speakers, films, mechanical lectures, pamphlets, exhibits and statistics," Dr. Snow said. "The volume of requests and inquiries has tripled since syphilis and gonorrhea became mentionables in normal conversation and in the public prints."

To organize the association's appeal for contributions and memberships, Dr. Snow announced that Donald C. Dougherty, of Cleveland, has just been appointed an associated director on the national headquarters' staff. Mr. Dougherty has been a specialist in institutional money-raising since 1920, with thirty campaigns for funds furnishing his experience for the social hygiene work. In 1927 he organized and managed the campaign which provided more than $8,000,000 for the University Hospitals in Cleveland.

Dr. R. H. Bishop, Jr., also of Cleveland, is chairman of the association's special finance committee. He was chairman of the $8,000,000 hospital campaign, and Mr. Dougherty was his assistant. He recently served as chairman and Mr. Dougherty as secretary of the Ohio committee of the association. Some of the Ohio activities are to be used as a pattern in other states, Dr. Snow said.

A DIETARY STUDY IN RHEUMATIC FEVER

SUE E. SADOW, A.B., JOHN P. HUBBARD, M.D., AND T. DUCKETT JONES, M.D.*

BOSTON

IT has been suggested in various medical reports that some error of diet may play a significant part in rheumatic fever. In view of the vast literature on nutrition and on rheumatic fever, however, it is surprising that so few real data concerning the interrelation of the two exist. The frequency with which rheumatic fever occurs in the industrial and poorer classes is largely responsible for the vague inferences that nutrition may in some way be significant in its etiology. Recent work concerning vitamin C deficiency in rheumatic fever is the first definite indication that diet may be an important factor. It has therefore seemed advisable to review for publication a dietary study conducted at the House of the Good Samaritan during the first 6 months of 1932. An attempt was made to give an optimal diet to patients with rheumatic fever in order to determine if there was any significant clinical improvement which was not shown by a control group on the usual hospital diet.

REVIEW OF THE LITERATURE

Vining[1] suggested that the rheumatism of childhood is based primarily on a nutritional disturbance which is brought about by a prolonged deficiency of vitamin B or of protein, with a relative excess of carbohydrate, or possibly by a combination of these factors. Thomson and Edgar[2] were unable to confirm Vining's hypothesis. These observers studied 22 rheumatic children, one half of the children receiving a high protein and the remainder a high carbohydrate diet. Each patient had a quart of milk daily. After 5 months, blood studies (pH, urea, nonprotein nitrogen and uric acid) showed no difference, either between the two groups or from the predietary studies. Thomson and Edgar concluded that diet was of no material importance in the etiology or course of rheumatic fever.

Warner and Winterton[3] analyzed separately the diets of rheumatic families and of the rheumatic children in these families. These diets were compared with the diets of children in poor-law institutions and in hospital groups of the same economic status. It was found that animal protein and dairy products were low in both the rheumatic subjects and the controls, and that the intake of carbohydrate was high. The intake of vitamin C in fresh fruit and vegetables was higher in the rheumatic children than in some of the control groups. They reported that no one dietary factor could be cited as a contributory cause of rheumatic fever.

Rinehart[4] and his co-workers have offered evidence to the effect that chronic scurvy with superimposed streptococcus infection in guinea pigs results in a histopathologic picture "strikingly similar" to that found in human beings with rheumatic fever. It was further suggested, as a result of epidemiologic and clinical data, that vitamin C deficiency may be a necessary and significant accompaniment of rheumatic fever. Stimson, Hedley and Rose[5] had difficulty in reproducing these changes in guinea pigs but succeeded after substituting for the living streptococci a toxin prepared from a scarlatinal strain of hemolytic streptococcus. The experimental results have, to some extent, been confirmed by Schultz,[6] but the changes were considered to resemble only slightly those seen in rheumatic fever. McBroom, Sunderland, Mote and Jones[7] demonstrated these changes in acute scurvy in the guinea pig and reported that the lesions were equally prevalent and severe in total scurvy irrespective of whether there was superimposed infection.

Faulkner[8] administered large daily doses of vitamin C to 27 patients with rheumatic fever, 8 with bone tuberculosis and 2 with Still's disease. There was no apparent influence on the clinical course of these patients. A reticulocyte response roughly proportional to the severity of the disease was noted. He suggested that during infection there is an increased demand for or utilization of vitamin C, which is not met by diets heretofore considered adequate.

Perry[9] studied the 12-hourly urinary output after the oral administration of 500 mg. of ascorbic acid in 5 active and 6 inactive rheumatic fever patients. Sendroy and Schultz[10] have made a much more extensive study of the urinary excretion test and have discussed the various factors affecting the results; 13 patients with active rheumatic fever, 13 with inactive rheumatic fever and 7 normals were studied. The results of both of the latter investigations fail to support the concept that a condition of vitamin C deficiency is a predisposing factor in the causation of rheumatic fever.

Of interest also is the recent report of Abbasy, Hill and Harris.[11] A striking difference was found between the range of values for the excretion of

From the House of the Good Samaritan, Boston. The expenses of this study have been defrayed by a grant from the Commonwealth Fund.

*Dietitian, Research Assistant, and Research Director respectively at the House of the Good Samaritan.

vitamin C in the urine of rheumatic patients (active and convalescent) and those of control subjects. The greatly increased metabolic use of and need for vitamin C and a correspondingly lowered degree of "saturation" of the body tissues were stressed. It is generally agreed from the work on vitamin C excretion that there is an increased utilization of this vitamin during infections. The claim of Abbasy, Hill and Harris that the urinary response is lowered in convalescence as well as in active rheumatic fever raises the question of the care needed in determining when rheumatic fever is quiescent. They have not given the criteria for this determination. It is therefore impossible to accept as proved the conclusion that rheumatic fever differs from any other infection in its vitamin C requirements.

Schultz,[12] in two comparable groups of rheumatic children, one of which received daily doses of ascorbic acid, found no evidence that the incidence of active rheumatic fever was favorably affected by the medication, although the development of subclinical scurvy was prevented in the treated group. Large doses of orange juice or ascorbic acid over periods of several months failed to affect demonstrably the clinical manifestations of active rheumatic fever. From this clinical study, Schultz stated that vitamin C deficiency is not a necessary factor in the etiology.

DIETARY STUDY

During the first 6 months of 1932 the children in this hospital were divided into two groups for the dietary study. To one group a special diet as described below was given; the second, which served as a control group, received the usual hospital diet. All of the children in both groups had well-established rheumatic fever, including certain cases where chorea was the only manifestation of their illness. The hospital is equipped to keep patients for long periods of time. The average length of stay for the experimental group was 35 weeks, for the control group 30 weeks. One patient included in the study was hospitalized for somewhat over 2½ years. In order to avoid so far as possible any transitory effects of the diet, only those children were included in this study who had been on either the experimental or the usual hospital diet for 10 weeks or longer. In the group on the experimental diet there were 31 such patients, 13 male and 18 female. These patients received the experimental diet for an average period of 20 weeks. This group included somewhat sicker children, since we wished to give them any benefits that might be derived from the optimal diet. The control group numbered 25 patients, 8 male and 17 female, with an average of 19 weeks during the dietary study. Throughout the 6 months of the experiment all the patients in both groups were weighed weekly. The average age for the experimental group was 9 years, with a range from 3½ to 15 years, and that of the control group was 10 years, with a range from 6 to 18 years.

The experimental diet was planned to supply optimal quantities of all known important food elements. No attempt was made to determine the effect of separate nutritional factors. This was arranged to include daily a quart of vitamin D milk, one egg, one serving of meat or fish, one potato, one other vegetable, a raw vegetable, such as lettuce, celery, cabbage or carrot, twice a day, 8 oz. of tomato or orange juice, with additional foods, such as bread, cereal, butter, and so forth, to make up the desired caloric content. Carefully measured portions were given, and throughout the study daily calculations, from the tables given by Rose[13] and Sherman,[14] were made on each serving of food for its contribution of calories, protein, fat, carbohydrate, calcium, phosphorus, iron and vitamins A, B complex, C and D. Combining these results, it was found that the average daily experimental diet offered during the 6 months of the study period contained the following:

Total calories	2,200-2,400
Grams:	
Protein	75
Fat	113-127
Carbohydrate	236-238
Calcium	1.4-1.7
Phosphorus	1.5-1.7
Iron	.012-.018

No attempt was made to estimate the vitamins quantitatively, but vitamins A and D were abundantly supplied by the quart of unpasteurized, irradiated vitamin D milk* and 4 oz. of cream, in addition to eggs and vitamin-containing yellow and green vegetables; vitamin B intake in food was supplemented by the daily addition of dried brewer's yeast, embo or bemax;† and 8 oz. of orange juice or tomato juice given daily supplied abundant vitamin C in addition to that contained in other fruits and vegetables. Ultraviolet irradiation by means of direct sunlight, ultraviolet lamp or a vita-glass enclosed porch supplied additional vitamin D. This latter was only for those children who were thought able to benefit from such irradiation.

The nutritive value of the usual hospital diet given to the control group was calculated in the

*This was kindly supplied by Walker-Gordon Laboratory Company.

†Bemax and embo are proprietary concentrates of vitamin B prepared from grain germ.

same manner as the experimental diet. The average daily contributions of this diet were as follows:

Total calories	1,600-1,800
Grams:	
Protein	64
Fat	86
Carbohydrate	203
Calcium	1.4
Phosphorus	1.4
Iron	.0085

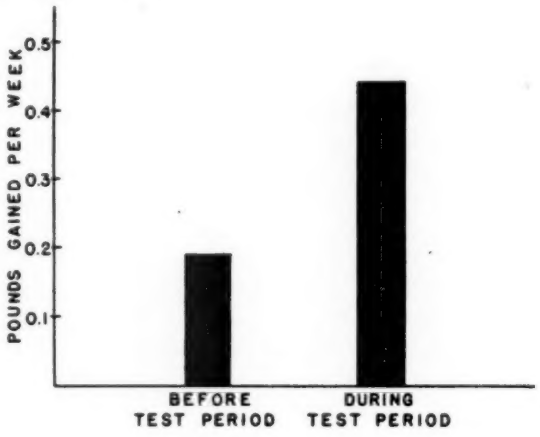
This diet included 10 cc. of cod liver oil daily. Other than this, no foods rich in vitamins were given.

It is obviously important in any dietary experiment to study not only the food offered but the quantity actually ingested. Throughout the entire study a careful record of food intake at each meal and general eating habits was kept for all the children in both groups. From a review of these records, every member of the two groups was classified according to whether he was a good eater consuming the entire diet offered him, or whether he was a fair, or a poor eater. This allowed approximate evaluation of comparable groups on the two diets.

RESULTS AND COMMENTS

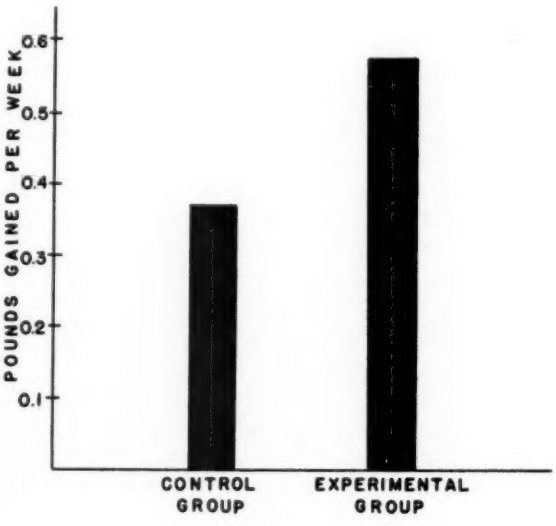
The most conspicuous result of the study was the gain in weight of the group on the optimal diet, which can be attributed to the 37.5 per cent greater caloric content of the diet. The average weekly gain for the total experimental group of 31 patients was 0.41 pounds. Of this whole group, 17 had been hospitalized for a period of 5 weeks or longer preceding the introduction of the experimental diet. The average gain per week for these 17 subjects during the period of their hospitalization preceding the introduction of the diet was 0.19 pounds, in comparison with an average gain of 0.44 pounds per week for the same 17 patients while on the experimental diet. Thus it is seen that the weekly gain for this group while on the diet was somewhat more than double that of the period when these same children were on the usual hospital diet (Chart 1). Chart 2 compares the test group with the control group. In order to make the two groups comparable, only those members of each group were included who were recorded as good eaters. There were 17 such patients in the experimental group, with a gain of 0.58 pounds per week. The control group of 22 good eaters gained 0.37 pounds per week. This comparison also shows a significant increased weight gain attributable to the optimal diet.

Aside from the gain in weight, we were unable to detect any significant change in the rheumatic process as a result of the diet. The children in the test group were for the most part sicker than those in the control group. They seemed to improve no more rapidly than might ordinarily be expected. Many of them had recurrences of various manifestations of rheumatic fever while on the experimental diet. There was no noticeable influence on the leukocyte count, the erythrocyte sedimentation rate or the duration of the P-R interval of the electrocardiogram.



GAIN PER WEEK FOR EXPERIMENTAL GROUP (17 PATIENTS) BEFORE AND DURING TEST PERIOD

CHART 1.



GAIN PER WEEK FOR CONTROL AND EXPERIMENTAL GROUPS DURING TEST PERIOD

CHART 2.

The height of all children was recorded near the beginning and end of the study period in order to determine whether there was any increased

growth for the test group in comparison with the control group. No significant difference was noted. If anything, the control group appeared to show a slightly greater increment than the test group.

That there should have been an increased rate of weight gain for the experimental group is not at all surprising considering the greater caloric content of the special diet. Nevertheless, we wish to point out that a significant gain in weight was brought about, even in spite of the continued activity of the rheumatic process, when special attention was paid to the diet. We are inclined to believe that, although according to present knowledge the nutritional state of an individual cannot be considered specific in prophylaxis or therapy, it is nevertheless an important factor in the individual's ability to combat disease. We agree with Swift[13] and McCulloch[16] that loss of weight or failure to gain may be the forerunner of a recurrence of rheumatic activity. Furthermore, it is of interest to note that gain in weight was one of the most striking features of the clinical improvement noted in children sent from Boston to Florida (Roche and Jones[17]) and in those sent from New York to Puerto Rico (Coburn[18]). Eventually it is to be hoped that the etiology of rheumatic fever will be conclusively proved so as to lead the way to a specific prophylaxis and treatment. Until that time, however, children suffering from rheumatic fever should receive a diet with ample caloric content and of high nutritional value. This is distinctly desirable in any chronic infectious disease.

SUMMARY

1. A diet intended to be optimal in all important nutritional elements was given to a group of children with rheumatic fever. The usual hospital diet was given to a similar group for control purposes. The diet of the experimental group had a 37.5 per cent higher caloric value than the diet of the control group.

2. There was a significant increase in the rate of weight gain for the group on the special diet. This occurred in spite of continued activity of the rheumatic process.

3. Other than gain in weight while on the diet of higher nutritional value, there was no observable difference in the course of the disease.

4. Since gain in weight appears to be desirable for the child subject to or suffering from rheumatic fever, the suggestion is made that more emphasis be placed on the need of a high caloric diet and one of high nutritional value.

REFERENCES

1. Vining, C. W.: The prerheumatic child. M. J. & Rec. **128**:351 (Oct. 3) 1928; 395 (Oct. 17) 1928; 453 (Nov. 7) 1928.
2. Thomson, A. P., and Edgar, S. H.: The effect of variation of diet in rheumatic children. Arch. Dis. Childhood **4**:152 (June) 1929.
3. Warner, E. C., and Winterton, F. G.: A dietetic study of cases of juvenile rheumatic disease. Quart. J. Med. **4**:227 (July) 1935.
4. Rinehart, J. F., and Mettier, S. R.: The heart valves and muscle in experimental scurvy with superimposed infection, with notes on similarity of lesions to those of rheumatic fever. Am. J. Path. **10**:61 (Jan.) 1934.
   Rinehart, J. F.; Connor, C. L., and Mettier, S. R.: Further observations on pathologic similarities between experimental scurvy combined with infection, and rheumatic fever. J. Exper. Med. **59**:97 (Jan.) 1934.
   Rinehart, J. F.: An outline of studies relating to vitamin C deficiency in rheumatic fever. J. Lab. & Clin. Med. **21**:597 (Mar.) 1936.
5. Stimson, A. M.; Hedley, O. F., and Rose, E.: Notes on experimental rheumatic fever. Pub. Health Rep. **49**:361 (Mar. 16) 1934.
6. Schultz, M. P.: Cardiovascular and arthritic lesions in guinea-pigs with chronic scurvy and hemolytic streptococcic infections. Arch. Path. **21**:472 (April) 1936.
7. McBroom, J.; Sunderland, D. A.; Mote, J. R., and Jones, T. D.: Effect of acute scurvy on the guinea-pig heart. Arch. Path. **23**:20 (Jan.) 1937.
8. Faulkner, J. M.: The effect of administration of vitamin C on the reticulocytes in certain infectious diseases; preliminary report. New Eng. J. Med. **213**:19 (July 4) 1935.
9. Perry, C. B.: Rheumatic heart disease and vitamin C. Lancet **2**:426 (Aug. 24) 1935.
10. Sendroy, J., Jr., and Schultz, M. P.: Studies of ascorbic acid and rheumatic fever; quantitative index of ascorbic acid utilization in human beings and its application to the study of rheumatic fever. J. Clin. Investigation **15**:369 (July) 1936.
11. Abbasy, M. A.; Hill, N. G., and Harris, L. J.: Vitamin C and juvenile rheumatism. Lancet **2**:1413 (Dec. 12) 1936.
12. Schultz, M. P.: Studies of ascorbic acid and rheumatic fever; test of prophylactic and therapeutic action of ascorbic acid. J. Clin. Investigation **15**:385 (July) 1936.
13. Rose, Mary S.: Laboratory Handbook for Dietetics. New York: Macmillan Co., 1930.
14. Sherman, H. C.: Chemistry of Food and Nutrition. New York: Macmillan Co., 1928.
15. Swift, H. F.: Factors favoring the onset and continuation of rheumatic fever. Am. Heart J. **6**:625 (June) 1931.
16. McCulloch, H.: Gain in weight as a guide in the convalescent care of cardiac children. Am. Heart J. **1**:569 (June) 1926.
17. Roche, C. F., and Jones, T. D.: Heart disease of the rheumatic type. J. Florida M. A. **20**:342 (Feb.) 1934.
18. Coburn, A. F.: The Factor of Infection in the Rheumatic State. Baltimore: Williams & Wilkins Co., 1931.

MASSACHUSETTS MEDICAL SOCIETY

PROCEEDINGS OF THE COUNCIL

Annual Meeting, June 2, 1937

THE annual meeting of the Council of the Massachusetts Medical Society was called to order by the president, Charles E. Mongan, of Somerville, in the Penthouse of the Hotel Bradford, Boston, on Wednesday, June 2, 1937 at 10:30 o'clock. There were 218 Councilors present; the list will be found in Appendix No. 1.

The report of the stated meeting of the Council, held on February 3, 1937, and published in the *New England Journal of Medicine* in the issue of March 25, 1937, was submitted by the Secretary and was accepted by vote. The Secretary next presented the record of the special meeting of the Council held on February 24, 1937, as published in the *New England Journal of Medicine* in the issue of April 15, 1937. The Council voted to accept the report as published.

The President directed the Secretary to read the following obituaries:

Dr. Robert Battey Greenough, of Brookline, Massachusetts, died at the Phillips House, Massachusetts General Hospital, on February 16, after a short illness, in his sixty-sixth year.

Born in Cambridge, he was graduated from Harvard College in 1892 and Harvard Medical School in 1896. He was appointed assistant professor of surgery, Harvard Medical School, in 1909 and visiting surgeon, Massachusetts General Hospital, in 1915, and held both positions until 1932 with the exception of his years in war service. He had been president of the Massachusetts Medical Society and the American College of Surgeons.

Dr. Greenough was always interested in the cancer problem and served on the Harvard Cancer Commission, at one time as chairman, and on the staff of the Collis P. Huntington Memorial Hospital, for many years as its head. At the time of his death he was president of the American Society for the Control of Cancer and a member of the Board of Managers, Memorial Hospital, New York.

He is survived by his widow and four daughters.

Dr. William Basil Keeler, of Roxbury, Massachusetts, died at his home February 11, 1937, after a short illness.

Born in Schenectady, New York, in 1877, Dr. Keeler came to Boston at an early age. He was a graduate of the Boston English High School and of Tufts College Medical School. Before taking up private practice, he served as an intern at the Boston City Hospital. From 1922 to 1928 he was medical inspector of the South Boston health unit and from 1928 to 1936 was in charge of the unit. He also assisted Dr. Charles F. Wilinsky, deputy commissioner in charge of child hygiene and director of the health units, from 1922 until 1936, and was supervising medical inspector of the parochial schools of Boston. In 1936 he was appointed health commissioner of Boston.

Dr. Keeler was a member of the Massachusetts Medical Society, the American Medical Association and the United States Public Health Association. He was a past president of the Norfolk District Medical Society and honorary president of the Boston Health League.

His widow and one daughter survive him.

Following the reading of the obituaries the Council stood in silence as a tribute to the deceased.

The Secretary called the roll of the Nominating Councilors and they withdrew from the meeting to consider the nomination of officers for the ensuing year. The President announced that the Nominating Councilor and the Alternate from the Worcester district were ill and unable to attend and, upon the suggestion of the president of the district society, the name of A. W. Marsh was presented. The Council voted to confirm Dr. Marsh. The President stated that the same condition had arisen in Berkshire district and presented the name of P. J. Sullivan to serve as Nominating Councilor. The choice was duly approved by vote. The Nominating Councilors were as follows:

W. D'A. Kinney, *Barnstable;* P. J. Sullivan, *Berkshire;* W. H. Allen, *Bristol North;* E. F. Cody, *Bristol South;* J. F. Burnham (Alternate), *Essex North;* J. F. Jordan (Alternate), *Essex South;* H. M. Kemp, *Franklin;* G. L. Schadt, *Hampden;* L. N. Durgin (Alternate), *Hampshire;* R. R. Stratton, *Middlesex East;* F. P. Murphy, *Middlesex North;* A. W. Dudley, *Middlesex South;* W. A. Griffin, *Norfolk;* C. A. Sullivan, *Norfolk South;* W. H. Pulsifer, *Plymouth;* F. H. Lahey, *Suffolk;* A. W. Marsh, *Worcester;* and A. A. Wheeler, *Worcester North.*

REPORTS OF STANDING COMMITTEES

Membership and Finance

Dr. David N. Blakely, Norfolk, presented the report of the Committee on Membership and Finance, which deals with membership. (See Appendix No. 2.) The Committee recommends that five Fellows be allowed to retire, that the dues of one Fellow be remitted, that four Fellows be allowed to resign, that ten Fellows be deprived of Fellowship for nonpayment of dues and that six Fellows be allowed to change membership from one district society to another without change of legal residence. The Council voted to accept the report and to approve of the recommendations.

Dr. Blakely stated that the Committee on Membership and Finance recommends that the surety bond of the Treasurer in the amount of $15,000 be renewed for one year from June 19, 1937. The

Council duly voted to approve the recommendation.

The President then declared the meeting to be in executive session and appointed Dr. Conley, Middlesex South, as sergeant-at-arms. The chairman of the Committee on Membership and Finance made a brief informal report, and since there was no objection the President granted the Committee's request to defer its formal report. The executive session was then dissolved.

Ethics and Discipline

The report of the Committee on Ethics and Discipline was read by Dr. Robert L. DeNormandie, Suffolk, in the absence of the chairman, Dr. Cheever, and was duly accepted by vote of the Council. (See Appendix No. 3.)

Medical Education and Medical Diplomas

Dr. Reginald Fitz, Suffolk, presented the report of the Committee on Medical Education and Medical Diplomas, which was accepted by vote of the Council. (See Appendix No. 4.)

State and National Legislation

The President gave a brief summary of the work of the Committee on State and National Legislation, stating that after a strenuous session the Committee can report practically one hundred per cent results. The chairman and other members of the Committee attended numerous hearings before legislative committees and supplied information and direction to the district legislative committees for their use. The Committee was surprised to have the legislature substitute the original bill for an adverse report in the case of the so-called Chiropractic Bill, but when the matter finally came to a vote (roll call) the measure was defeated. No legislative counsel was employed, which resulted in a considerable saving to the Society. The Council was warned that still closer contact must be established between the individual members of the profession and the members of the legislature if the medical viewpoint on various legislative matters is to be thoroughly understood. Dr. Mongan spoke highly of the receptive attitude shown by the majority of the legislators and the courtesy with which the Society's representatives were treated by committees. He also spoke concerning the co-operation which the Committee received from members of the Society.

Public Health

In the absence of the chairman of the Committee on Public Health and the Subcommittee on Public Education the report was presented by Dr. Marsh, Worcester, and was duly accepted by the Council. (See Appendix No. 5.)

Malpractice Defense

Dr. Franklin G. Balch, Norfolk, presented the report of the Committee on Malpractice Defense, which contained recommendations to bring about changes in the by-laws and in the Malpractice Defense Act. The report was accepted and the recommendations approved. (See Appendix No. 6.) As a result the necessary changes in by-laws and in the defense act will be acted upon by the Society at its annual meeting on June 3.

Permanent Home

Dr. William H. Robey, Suffolk, presented the report of the Committee on Permanent Home, which was duly accepted by vote. (See Appendix No. 7.)

Publications

Dr. Roger I. Lee, Suffolk, chairman of the Committee on Publications, made an informal report for his Committee stating that the editorship of the *New England Journal of Medicine* had been transferred from Dr. Bowers to Dr. Nye. He stated that the Committee would be glad to receive suggestions and criticisms. The Council voted to accept Dr. Lee's report.

REPORTS OF SPECIAL COMMITTEES

The Secretary presented the reports of the committees previously approved by the Council and charged with the duty of investigating petitions for restoration. All reports being favorable, the Council voted to restore the applicants listed below to Fellowship under the conditions specified in each case.

1. N. M. Baghdoyan, Arlington. (Committee: R. W. Sheehy, M. J. Quinn and J. H. Blaisdell.)
2. Roger E. Hubbard, Greenfield. (Committee: A. H. Ellis, A. E. Johnson, Jr. and H. M. Kemp.)
3. Herbert R. Toombs, Westfield. (Committee: A. J. Douglas, H. B. Chase and R. D. Hildreth.)
4. Clifton L. Buck, Beverly. (Committee: P. P. Johnson, A. E. Parkhurst and R. E. Alt.)

The President nominated committees to consider petitions for restoration to Fellowship submitted by certain Fellows who had previously been deprived. The nominations were duly approved by the Council and are listed in Appendix No. 8.

Cancer

Dr. Shields Warren, Suffolk, presented the report of the Committee on Cancer, which was duly accepted by vote. (See Appendix No. 9.)

Postgraduate Instruction

Dr. Frank R. Ober, Suffolk, presented the report of the Committee on Postgraduate Instruction, which was duly accepted by the Council. (See Appendix No. 10.) There then followed some explanatory discussion of the report from which it appeared that all registered physicians in the State are eligible to receive certain postgraduate instruction provided under the Social Security Act. The Council was informed that the Commissioner of Public Health had asked the Massachusetts Medical Society, through this Committee, to assist in the undertaking. The Council finally voted to accept the recommendations of the Committee as follows:

1. That the Committee be authorized to co-operate with the state and federal authorities in providing postgraduate instruction to the medical profession in the Commonwealth and
2. That the Committee be continued in order to carry out the above co-operation and to assist in the preparation of a plan.

Physical Therapy

The report of the Committee on Physical Therapy was presented by the chairman, Dr. Franklin P. Lowry, Middlesex South, and was duly accepted by the Council. (See Appendix No. 11.)

Public Relations

Dr. Elmer S. Bagnall, secretary of the Committee on Public Relations, presented a brief report of the activities. (See Appendix No. 12.) Dr. Lane, Norfolk, raised a question regarding certain matters in connection with the prepayment hospital plan. The chair finally ruled, however, that the material submitted by Dr. Lane constituted new business and the Council proceeded to approve of Dr. Bagnall's report.

Dr. J. Harper Blaisdell, Middlesex East, chairman of the Subcommittee on Hospital Relations of the Committee on Public Relations, stated that he had been charged by the Committee on Public Relations with the preparation of information vital to the proper handling of industrial accident cases and that under date of May 27, 1937, this information had been duly published in the *New England Journal of Medicine* and reprints of the article had been distributed prior to the Council meeting. Dr. Blaisdell amplified the printed report somewhat and answered certain questions which were raised. Dr. Blaisdell's report in effect was the statement entitled "Compensation of physicians in industrial cases," published in the *New England Journal of Medicine* as stated above. The Council voted to accept the report and to approve of the recommendation that it be sent in reprint form to the Fellows of the Massachusetts Medical Society and to such other persons as may be properly concerned.

Gentleman's Agreement

Dr. William G. Curtis, Norfolk South, presented a report of a special committee which had been appointed to co-operate with similar committees from hospital and insurance groups and which had been largely instrumental in negotiating an informal agreement which it is hoped will expedite claims of physicians for professional services rendered in automobile accident cases. (See Appendix No. 13.) The work originally started in connection with the district medical societies adjacent to Boston but has now been so planned as to permit of its extension throughout the state. The original agreement was published in full in the *New England Journal of Medicine,* issue of March 25, 1937. The original forms for submitting claims have been slightly modified. Dr. Curtis moved the acceptance of the report. This was duly voted by the Council. The recommendation regarding the alteration of the forms was next presented and duly adopted.

Miscellaneous

A special committee, of which Dr. Lincoln Davis, Suffolk, is chairman, presented the following resolution:

The Council of the Massachusetts Medical Society desires to record its deep appreciation of the services to this Society of Dr. Walter P. Bowers upon his retirement as managing editor of the *New England Journal of Medicine.* Dr. Bowers's services have been manifold and notable, in his own community, in his district medical society, and as President and Councilor of the Massachusetts Medical Society. His untiring labors in behalf of higher medical standards and better medical service, as a member of important committees, and on the Board of Registration in Medicine of the Commonwealth of Massachusetts have been of inestimable value. His sound judgment, courage, and devotion to duty have always been found available in the furtherance of every good cause.

When the Massachusetts Medical Society took over the *Boston Medical and Surgical Journal,* Dr. Bowers became its editor. The phenomenal success of the *Journal,* its influence in medical affairs and medical education, not only in Massachusetts but throughout New England, has been the work of Dr. Bowers. As editor of the *Journal,* he has been modest and unassuming, but at the same time he has been an uncompromising idealist, an indefatigable worker and a most devoted servant of the medical profession and of the Massachusetts Medical Society.

The Council of the Massachusetts Medical Society takes pleasure in making grateful acknowledgment of the valued services of Dr. Bowers, secure in its belief that his example will furnish an inspiration to future editors for many years to come.

ROGER I. LEE,
ARTHUR W. MARSH,
LINCOLN DAVIS.

Dr. Davis moved the adoption of the resolution and it was unanimously carried.

Dr. John M. Birnie, Hampden, as chairman of a committee appointed by the President to study the present boundaries of the district societies of the Commonwealth, reported progress.

A committee of which Dr. Philip H. Cook, Worcester, was chairman presented a report on the matter of information to be included in directories. (See Appendix No. 14.) It was duly accepted by the Council.

Dr. Walter P. Bowers, Worcester, chairman of the committee appointed to draw up a suitable resolution recognizing the services rendered by the late Dr. Robert B. Greenough, read the following resolution which had been prepared by his committee:

RESOLUTION ON THE DEATH OF DR. ROBERT BATTEY GREENOUGH

On February 16, 1937 Dr. Robert Battey Greenough, a Fellow of the Massachusetts Medical Society, died, aged 65 years, 3 months and 9 days.*

After an elaborate premedical education and graduation from the Harvard Medical School and the Massachusetts General Hospital, Dr. Greenough engaged in postgraduate studies abroad and on returning to Boston entered upon the practice of medicine, selecting surgery as his specialty.

Naturally endowed with unusual qualities of mind and heart and proceeding in orderly sequence through the steps of a professional career, he reached a commanding position in the Massachusetts Medical Society and the several scientific organizations to which he was elected.

After having established a nation-wide influence in his specialty, he became a leading figure in public activities relating to the instruction of the medical profession and the laity respecting the problems presented by cancer.

It is fitting that the Massachusetts Medical Society should place on record its profound sense of loss in the death of our esteemed Fellow, who served as president in the years 1929, 1930 and 1931. A notable feature of his official functions was the impressive celebration of the one hundred and fiftieth anniversary of the founding of the Society. His work on many committees of the Society was constructive especially in the field of legislation and in plans for a permanent home for the Society.

Because of his devotion to his patients he was beloved by all who came under his ministrations.

Therefore, it is hereby

RESOLVED: That in the death of Dr. Robert Battey Greenough, the Massachusetts Medical Society has lost a devoted and inspiring leader, and that our sympathy is hereby extended to his widow and children; and further that copies of this resolution shall be spread on the records of the Society and sent to Dr. Greenough's family.

WALTER P. BOWERS,
CHARLES S. BUTLER,
FRANKLIN S. NEWELL.

The Council rose in confirmation of the recommendations of the Committee.

The President stated that at the last Council meeting he was authorized to appoint a committee whose duty it should be to consider and make recommendations regarding the committee work of the Society. He stated that on this committee he would appoint as members the chairmen of the various standing committees with the president of the Society as chairman.

Nominating Committee

The Nominating Committee, through its chairman, Dr. Cody, Bristol South, announced its selections for the various offices in the Society as follows:

For president: Channing Frothingham, Boston.
For vice-president: Walter G. Phippen, Salem.
For secretary: Alexander S. Begg, West Roxbury.
For treasurer: Charles S. Butler, Boston.
For orator: Allen G. Rice, Springfield.

The President announced the above nominations and asked if there were other nominations from the floor, and there being none he declared the nominations closed. On motion of Dr. Lane, duly seconded, the Council voted to suspend the rules and to direct the Secretary to cast one ballot for the list of names submitted by the Nominating Councilors. The Secretary announced that the ballot had been cast and that the above-named officers were duly elected.

The President welcomed the President-Elect to the platform, where Dr. Frothingham made a few appropriate remarks.

The President announced the standing and special committees for the ensuing year. (The list is to appear in the Proceedings of the Society, soon to be published.) The appointments were confirmed in regular manner by vote of the Council.

The President announced that he was presenting for confirmation the name of Elmer S. Bagnall, of Groveland, secretary of the Committee on Public Relations and secretary of the Essex North District Medical Society, to be a delegate to the House of Delegates of the American Medical Association. The Council voted to approve the appointment.

NEW BUSINESS

The Secretary presented a petition from certain Fellows of the Society who are also members of the New England Physical Therapy Society requesting that the Council authorize the separation of the Section of Radiology and Physiotherapy into two sections, and that the name "Physiotherapy" be changed to "Physical Medicine." The petition was signed by fourteen men. Dr. Shields Warren, Suffolk, expressed the hope that the recommendation would not be favorably acted upon since he

*For a brief record of Dr. Greenough's life see the *New England Journal of Medicine* of March 11, 1937.

deplored the tendency to separate in a formal way the various branches of medicine.

Dr. Cook, Worcester, speaking for the Fellows of the Society who are also members of the New England Roentgen Ray Society, likewise presented a petition asking for division of the section. The petition was signed by thirty-six men.

Dr. Robey, Suffolk, pointed out that the division of the section would result in there being eight sections of the Massachusetts Medical Society, which in his opinion was too large a number. He felt that the members would get more information at the general meetings, but that because of the existence of so many sections they were unable to attend. He stated that he was opposed to the formation of another section.

Dr. Helen S. Pittman, Suffolk, reported that the Section of Tuberculosis had recently voted to disband since they wished to go back to the Section of Medicine.

After some discussion it was finally voted to refer the entire matter to the Committee of Arrangements and ask it to report at a future meeting. Since there was some doubt about the voice vote, a count was made which showed 93 favoring the motion and 63 opposed. The President announced that the matter would be referred to the Committee of Arrangements.

Dr. Levi, Middlesex South, stated that inasmuch as a resolution previously submitted by him had been found to conflict with the by-laws, he now desired to submit another resolution as follows:

> Resolved: That the names and addresses of applicants for membership in the Massachusetts Medical Society and the name and address of the secretary of the district be published together in a special list in the *New England Journal of Medicine* at least two weeks prior to each censors' meeting.

After some explanatory discussion the Council voted to adopt Dr. Levi's resolution.

Dr. Lane's motion, previously made, was reread by the President. It was as follows:

> The Massachusetts prepaid hospitalization plan shall exclude all professional or medical services of physicians or surgeons, excepting salaried pathologists, roentgenologists, anesthetists and special nurses.

After considerable discussion Dr. Blaisdell rose to a point of order, namely, that the motion looks toward compulsory action upon a now existing Massachusetts corporation over which the Council has no jurisdiction. He stated that the Committee on Public Relations had no motive to keep out discussion on the problem and if the present motion is ruled out of order he would make a motion that it is the sense of the meeting that payment for any anesthesia services should be excluded. The President confirmed the attitude of Dr. Blaisdell and ruled Dr. Lane's motion out of order. Dr. Blaisdell then made the following motion:

> That it is the sense of this meeting that the Associated Hospital Service Corporation of Massachusetts exclude from the provisions of its policy all payment for the services of anesthetists.

The motion was duly seconded and, after it was reread for the better understanding of the members, Dr. Blaisdell stated that he wished to speak in opposition to the adoption of the motion. Dr. Blaisdell went on to state that the Council had approved in principle a prepayment hospital insurance plan and that it was left to the public relations committee to adjust the variety of difficulties which would arise. This was done with the exception of that regarding the payment for anesthetists. The original policy suggested the inclusion of services of anesthetists and, on the advice of the Committee on Public Relations, the wording was changed so that the clause now reads "that the cost of anesthesia to a maximum of ten dollars will be paid by the corporation." The anesthetists were represented by the Boston Anesthesia Society with a membership of about twenty-five men, some of whom have stated that they are unalterably opposed to having the cost of anesthesia included. It is at least debatable how far these specialists represent the many general practitioners who in the course of their work are giving anesthesia and who might welcome a guarantee toward the cost of anesthesia to a maximum of ten dollars. The public relations committee has discussed this question at great length and the conclusion, though not unanimous, is that the cost of anesthesia to a maximum of ten dollars should be left in at this time. The policy is not unalterable, and the light of experience may teach us that changes in the future will be necessary. He expressed the hope that the motion would be defeated. The Council then proceeded to vote on the question and Dr. Blaisdell's motion was lost.

The President announced the contemplated changes in the by-laws which had been sent out with the call of the meeting and which are to be acted upon at the annual meeting of the Society on June 3. The various provisions were presented seriatim and each was individually approved by formal vote.

The President closed the meeting by extending his thanks to the members of the Council for their faithful attendance and for the interest shown in the work. He expressed his appreciation of the

co-operation given him, and stated that in his opinion the Society should constitute a great influence in the community for proper legislation which affects the health in the community.

The meeting adjourned at 1:45 p. m.

ALEXANDER S. BEGG, *Secretary.*

APPENDIX NO. 1

ATTENDANCE

BARNSTABLE
M. E. Champion
W. D'A. Kinney

BERKSHIRE
G. P. Hunt
I. S. F. Dodd
John Hughes
P. J. Sullivan

BRISTOL NORTH
H. L. Rich
W. H. Allen
A. R. Crandell

BRISTOL SOUTH
J. A. Barre
G. W. Blood
R. B. Butler
E. F. Cody
E. D. Gardner
H. E. Perry
I. N. Tilden
C. C. Tripp

ESSEX NORTH
W. D. Walker
E. S. Bagnall
R. V. Baketel
C. S. Benson
J. F. Burnham
Z W. Colson
H. F. Dearborn
H. R. Kurth
L. C. Peirce
G. L. Richardson
F. W. Snow
C. F. Warren

ESSEX SOUTH
N. P. Breed
C. L. Curtis
J. F. Donaldson
R. E. Foss
J. F. Jordan
B. B. Mansfield
A. E. Parkhurst
C. H. Phillips
W. G. Phippen
J. W. Trask

FRANKLIN
H. M. Kemp
H. G. Stetson
A. H. Wright

HAMPDEN
A. G. Rice
F. H. Allen
T. S. Bacon
E. P. Bagg, Jr.
J. M. Birnie
L. D. Chapin
J. L. Chereskin
A. J. Douglas
Frederic Hagler
G. D. Henderson
E. A. Knowlton
M. W. Pearson
G. L. Schadt
H. L. Smith
G. L. Steele

MIDDLESEX EAST
L. M. Crosby
J. H. Blaisdell
Richard Dutton
E. M. Halligan
J. H. Kerrigan
K. L. Maclachlan
R. R. Stratton
E. E. Tyzzer

MIDDLESEX NORTH
F. D. Lambert
M. L. Alling
D. J. Ellison
A. R. Gardner
F. P. Murphy
M. A. Tighe

MIDDLESEX SOUTH
F. R. Jouett
C. F. Atwood
E. W. Barron
C. F. K. Bean
E. H. Bigelow
G. F. H. Bowers
B. F. Conley
D. F. Cummings
H. F. Day
J. E. Dodd
A. W. Dudley
H. Q. Gallupe
H. G. Giddings
H. W. Godfrey
W. G. Grandison
A. D. Guthrie
F. A. Higginbotham
N. M. Hunter
A. M. Jackson
A. A. Levi
F. P. Lowry
R. A. McCarty
F. F. McGirr
J. A. McLean
Edward Mellus
C. E. Mongan
J. P. Nelligan
E. J. O'Brien, Jr.
Dwight O'Hara
Max Ritvo
E. S. A. Robinson
E. J. Sawyer
M. J. Schlesinger
E. F. Sewall
E. W. Small
F. G. Smith
H. P. Stevens
R. A. Taylor
Fresenius Van Nüys
R. H. Wells
M. W. White
W. S. Whittemore

NORFOLK
Frederick Reis
F. G. Balch
A. S. Begg
M. G. Berlin
M. I. Berman
D. N. Blakely
Myrtelle M. Canavan
A. H. Ehrenfried
D. G. Eldridge
H. M. Emmons
C. B. Faunce, Jr.
J. F. Ford
L. M. Freedman
Eli Friedman
Maurice Gerstein
W. A. Griffin
J. B. Hall
L. F. Johnson
G. W. Kaan
C. J. Kickham
E. L. Kickham
H. M. Landesman
W. A. Lane
J. S. H. Leard
D. L. Lionberger
F. P. McCarthy
W. H. McMann
H. C. Petterson
Cadis Phipps
E. P. Ruggles
M. V. Safford
D. D. Scannell
J. A. Seth
F. J. Simmonds
H. F. R. Watts
S. H. Weiner
G. W. Winchester

NORFOLK SOUTH
N. R. Pillsbury
C. S. Adams
D. J. Bailey
W. G. Curtis
G. V. Higgins
F. E. Jones
C. A. Sullivan

PLYMOUTH
L. A. Alley
Jacob Brenner
H. A. Chase
A. L. Duncombe
W. H. Pulsifer

SUFFOLK
Conrad Wesselhoeft
A. W. Allen
G. M. Balboni
Walter Bauer
H. L. Blumgart
W. B. Breed
C. S. Butler
Lincoln Davis
R. L. DeNormandie
G. B. Fenwick
Reginald Fitz
Channing Frothingham
Joseph Garland
Rudolph Jacoby
E. P. Joslin
F. H. Lahey
R. I. Lee
C. C. Lund
L. S. McKittrick
J. P. Monks
N. A. Nelson
R. N. Nye
F. W. O'Brien
J. P. O'Hare
L. E. Parkins
Helen S. Pittman
G. P. Reynolds
W. H. Robey
Horatio Rogers
G. C. Shattuck
R. M. Smith
E. F. Timmins
I. J. Walker
Shields Warren
C. F. Wilinsky

WORCESTER
W. A. Bryan
J. C. Austin
W. P. Bowers
L. R. Bragg
P. H. Cook
G. A. Dix
E. B. Emerson
G. E. Emery
J. M. Fallon
J. J. Goodwin
E. L. Hunt
W. F. Lynch
E. C. Miller
W. C. Seelye
R. J. Ward
F. H. Washburn
S. B. Woodward

WORCESTER NORTH
E. A. Adams
H. C. Arey
A. F. Lowell
H. R. Nye

APPENDIX NO. 2

REPORT OF THE COMMITTEE ON MEMBERSHIP AND FINANCE ON MEMBERSHIP

This Committee recommends:

1. That the following named five Fellows be allowed to retire under the provisions of Chapter I, Section 5, of the by-laws:

Gray, Alice Maud, Roxbury.
McLaughlin, Joseph Ignatius, Roxbury.
Morris, Richard Holt, Everett, with remission of dues, 1937.
Piper, Frank, Boston.
Stacey, Charles Franklin, Boston.

2. That dues of the following named Fellow be remitted under the provisions of Chapter I, Section 6, of the by-laws:

Kramer, Florence Radinoff, Lynn, 1934 (in part), 1935, 1936.

3. That the following named four Fellows be allowed to resign under the provisions of Chapter I, Section 7, of the by-laws:

Claiborne, Thomas Sterling, Atlanta, Ga., with remission of dues, 1937.
Kobes, Herbert Richard, Augusta, Me., with remission of dues, 1937.
Macrae, Annie Campbell, Eldon, P. E. I., with remission of dues, 1937.
Reed, William Page, New York City, with remission of dues, 1937.

4. That the following named ten Fellows be deprived of the privileges of Fellowship under the provisions of Chapter I, Section 8, Clause (a) of the by-laws:

Clement, Merton Wallace, Worcester.
Creager, Florence Isabelle, Woburn.
Green, Abraham, Brookline.
Hunter, William Andrew, East Gardner.
Lopatin, Edward, Worcester.
Lugitch, Maurice, Dorchester.
Murray, Patrick Joseph, Dorchester.
Scott, George Henry, Roxbury.
Sternstein, Herman Jerry, Brookline.
Zervas, Themistocles Nicholas, Lynn.

5. That the following named six Fellows be allowed to change their membership from one district society to another without change of legal residence, under the provisions of Chapter III, Section 3, of the by-laws:

Two from Middlesex South to Suffolk

Marble, Henry Chase, Newton Center.
Merritt, Hiram Houston, Cambridge.

Three from Norfolk to Suffolk

Clute, Howard Merrill, Chestnut Hill (Brookline).
Fulton, Marshall Nairne, Brookline.
Henderson, Francis Freeman, Milton.

One from Suffolk to Norfolk

Praglin, Addy, Boston.

DAVID N. BLAKELY, *Chairman.*

APPENDIX NO. 3

REPORT OF THE COMMITTEE ON ETHICS AND DISCIPLINE

During the year just closing six formal meetings of the Committee have been held, in addition to the usual conferences between the chairman and individual Fellows and laymen. During the chairman's absence on account of illness for several months, his place was filled by the senior member of the Committee, Dr. R. L. DeNormandie.

It is with the greatest regret that the resignations of three members of the Committee, Drs. Warren D. Ruston, Sylvester F. McKeen and Alfred C. Smith, are reported. These gentlemen had served for many years, and contributed devoted and interested service, fair and disinterested judgment, and a liberal and enlightened point of view, each according to his experience, to the solution of many difficult problems. It is with especial and very personal sorrow that the subsequent death of Dr. McKeen must be recorded.

The resignation of a Fellow from the Society was demanded and obtained on the ground that he had given grossly improper testimony in court as an expert witness. Another Fellow was recommended to the President for severe admonition, for an offense of the same nature but of less serious degree.

A hearing was accorded a Fellow whose license to practice had been suspended for six months on account of gross negligence in the performance of an operation, and who had engaged in practice after his suspension. Action was postponed pending the trial of a suit for malpractice.

A serious charge against a Fellow, that he is using nostrums and secret remedies and making fraudulent claims for their efficacy, is now under investigation.

A number of instances of alleged exorbitant fees, of advertising, and of minor offenses were considered and appropriately disposed of, without invoking formal discipline. In all, the alleged misdemeanors of twenty-one Fellows were investigated. When called upon, the Committee has given advice about the ethical propriety of some course of action, to Fellows or medical organizations.

Two particular aspects of the work, always of concern, have attracted especial attention. One is the question of what violations of the Code of Ethics shall be held to deserve discipline, and how that discipline may be made prompt and effective. It is evident and reasonable enough that the well-nigh universal change, relaxation and even demoralization of previously accepted standards of viewpoint and behavior, so evident in the community at large, should have affected the medical profession. Such offenses as unethical treatment of brother physicians, dishonesty, exorbitant fees, misleading or even false testimony as an expert witness, seem to be definitely on the increase. The severe economic pressure and competition to gain a livelihood in our overcrowded profession throw temptation in the way of the weak.

It is evident that if membership in the Massachusetts Medical Society is to continue to be a guarantee, as it has been in the past, of reasonably high professional attainments, dependability, and moral integrity, these violations of our code must be met by effective disciplinary measures. It seems as though the present machinery were inadequate and cumbersome.

The Committee has two methods at its disposal: first, a recommendation for admonition by the President; secondly, the bringing of the accused before a board of trial, which may in its turn recommend admonition or expulsion. The former seems but a mild measure, appropriate only for trivial cases. The second is, it is true, severe, but the machinery involved is cumbersome and by no means efficient. The necessity of referring the verdict of the board of trial to the annual meeting of the Society in June often involves long delay, and since the number attending the annual meeting is but a very small fraction of the total membership; and since those in attendance

have no accurate knowledge of the case before them, their action is not likely to add to the probability of the attainment of justice. It is true that the Committee has the right, under the by-laws, to ask for a resignation, but no power to enforce the request; nevertheless, this provision has been of great value. Perhaps other disciplinary measures less lenient than admonition and less severe than expulsion should be provided for. Such, for instance, as suspension of Fellowship for a certain period. Would it not be wise also for the board of trial to report to the next meeting of the Council, which would be empowered to give final effect to the verdict? The Council is a deliberative executive body whose meetings are always fully attended and which thus truly represents the Society at large, and which can be made more easily informed of the facts of a case than can such a gathering as the annual meeting. At any rate, some measures to increase the certainty and celerity of disciplinary measures would seem to be advisable.

The other phase of the work of the Committee which, as heretofore, continues to be puzzling, is that of publicity of the activities of members of the profession which may reasonably be interpreted as unethical advertising. The question is a broad and difficult one, impossible of full discussion here. Shall the medical profession strive to maintain the long-accepted concept of the honorable physician as a man who avoids anything which savors of self-advertisement, who does not permit his professional activities or attainments to be broadcast either in the press or by radio, and who believes that the only ethical method of advertisement is by the good reports of his patients and by his participation in the activities of gatherings of his medical brethren and in communications to the medical press? Tremendous pressure is brought on the physician to yield to the importunities and alleged necessities of modern publicity—the usual alleged object of which is the "education" of the laity in medical matters. The physician is urged to permit the publication of medical articles in the lay press. He is invited to speak on medical topics before lay audiences of all kinds. He is asked to broadcast the same subjects by radio. Moreover, it appears that in this country, at least, he must yield to the requirements of the press and radio that all such communications shall be over his personal signature and often, in the case of the press, that they be accompanied by a photograph. By no means all these publicity stunts are genuinely useful and in the public interest; on the other hand, they unquestionably furnish a most effective advertisement of a physician, which quite naturally leads to envy and dissatisfaction on the part of others, who believe, often with justice, that he is enjoying undue advantage over them. All of this publicity opens the door to genuinely unethical self-advertisement by certain members of the profession; and the Committee finds it increasingly difficult to justify its critical attitude toward these men who point to the activities of their more favored colleagues as similar in their results in the matter of self-advertisement, even though approved by authority.

The Committee feels that this publicity is an entering wedge which may deprive the profession of medicine of one of its most characteristic and cherished attributes—a dependence on deeds rather than on words to ensure recognition and confidence. This matter of publicity is far too big to be settled for the Society by the Committee on Ethics and Discipline. It must receive the careful consideration of the district societies, and any important change in our present concept of the ethical principles involved should be carefully studied and approved by the whole Society.

DAVID CHEEVER, *Chairman.*

APPENDIX NO. 4

REPORT OF THE COMMITTEE ON MEDICAL EDUCATION AND MEDICAL DIPLOMAS

During the past year this Committee has held four meetings, at two of which candidates from schools not recognized by the Society have been examined. Such candidates continue to be a considerable problem for this Committee; the situation is now complicated by the fact that each year there are a few candidates who have come to this country from excellent European schools. The question arises as to how graduates from good foreign medical schools should be received by the Massachusetts Medical Society. On the whole, it has been so far the policy of your Committee to interpret the phrase in Chapter VII, Section 5, of the by-laws, "when such a diploma is presented by an applicant who has practiced for a minimum of five years," as meaning a minimum of five years in this country. We have adopted this policy deliberately because of the confusion that is known to exist regarding the admission to this country of medical men with a foreign education, and it has seemed wise to the Committee at the present time to make it a little difficult for foreigners to acquire the privileges of the Massachusetts Medical Society until such foreigners have been long enough established in the Commonwealth to make sure that they are desirable adjuncts. In certain instances this policy has entailed a hardship on some excellent, well-trained men of high reputation. On the other hand, the Committee will continue to follow the policy mentioned, unless it receives instructions from the Council to do otherwise, until the status of the foreign-trained medical men in this country is more clearly established.

The Committee has held two additional meetings for discussion of its general work. During the past year the American Medical Association has inspected the various hospitals approved for intern training in Massachusetts and has been kind enough to forward to your Committee the result of its studies. Your Committee plans to abstract and analyze these inspections with the idea of reviewing the matter of intern training in Massachusetts, feeling that the period of internship is one of the most important aspects of medical education that now exists.

At the meeting of the Council on February 5, 1936, Dr. A. A. Levi, Middlesex South, presented the following resolution:

> RESOLVED: That the names and addresses of applicants for membership in the Massachusetts Medical Society, together with the names of members of the Society acting as sponsors in such cases requiring this action (in accordance with the by-laws of the Massachusetts Medical Society, Chapter I, Section 1, and Chapter V, Section 1, and Chapter VII, Section 5) and the name and address of the secretary of each district be published in the *New England Journal of Medicine* three weeks prior to each Censors' meeting.

At a meeting of the Council on October 7, 1936, it was voted to refer this matter to the Committee on Medical Education and Medical Diplomas.

The Committee has conferred with Dr. Levi. The Committee feels that the sense of this resolution is excellent; as the resolution is worded, however, its meaning is not altogether clear and is capable of misinterpretation. On this account the Committee recommends to the Council that Dr. Levi's resolution in its present form be not adopted.

REGINALD FITZ, *Chairman.*

APPENDIX NO. 5

Report of the Committee on Public Health and the Subcommittee on Public Education

There has been no meeting and there is no report for the Committee on Public Health.

The Subcommittee on Public Education has been active, though there has been but one formal meeting.

For several years there has been a weekly health broadcast sponsored jointly by the Subcommittee on Public Education of the Massachusetts Medical Society and the Massachusetts Department of Public Health.

During most of this time the chief function of the Subcommittee on Public Education has been to review the broadcasts and approve them or otherwise, the broadcasts themselves having been arranged for by the Department of Public Health. The latter has done most excellent service in this matter and has made every effort to keep a high standard.

This committee has continued these health broadcasts weekly over station WBZ until the last of April, when they were changed to WAAB. This was because WBZ had decided to use the broadcasts of the American Medical Association as they were doing in most other places, and it was necessary for us to find some other station. Our broadcasts will be continued over WAAB on Thursday afternoons at 1.15 o'clock until the end of June.

During the year the question has come up and been given much consideration by a subcommittee as to whether these broadcasts were worth while and what could be done to improve them. These questions have not been studied exhaustively by any means, but it appears that, estimating from letters received from listeners on the basis used by broadcasting companies for this purpose, our average audience was probably about a quarter of a million.

The only suggestion for improvement of the broadcasts which has thus far been tried out is that members of the Committee be each personally responsible for a broadcast.

This has been done during the last few months, and I feel that both the subjects and the quality of the broadcasts have been greatly improved.

The list of topics for the next year has been prepared, but the speakers have not been selected.

Royal P. Watkins, *Chairman.*

APPENDIX NO. 6

Report of the Committee on Malpractice Defense

This year we must make a change in our Medical Defense Act. Our attorneys tell me that the Supreme Court of the State has decided in a similar case that as at present worded it amounts to the Society's practicing law, and that is not allowed. The slight changes made in the wording of the leaflet enclosed in the notices of the annual meeting seem to our attorneys sufficient to make the act legal. The Committee suggests substituting the following defense act in place of the present one:

Proposed Malpractice Defense Act

Active fellows of the Massachusetts Medical Society, in the event that they are sued for malpractice or for alleged illegal acts in connection with the commitment of persons to institutions for the insane, shall be entitled, on conditions hereinafter specified, to have the Society pay for the services of their attorneys in connection with the defense of such suit.

Such benefit shall be available on the following conditions:

1. An active member of the Society desiring to avail himself of the privileges of this act shall make application therefor in writing to the secretary of the medical defense committee, and shall show to the satisfaction of said committee that he is a Fellow in good standing in the Society, and was at the time of the alleged malpractice, and that all of his pecuniary obligations to the Society by way of dues and assessments have been duly discharged. He shall also furnish to the secretary of the committee upon request a complete and accurate statement of his connection with and treatment of persons upon which the suit against him is based, giving dates of attendance, names and residences of physicians, nurses and other persons having knowledge of facts and circumstances necessary to a clear and definite understanding of all matters in question, and shall furnish such other relevant information, if possible, as may be required of him by the secretary of the said committee.
2. In the event that the Committee on Medical Defense shall decide that the applicant is entitled to the benefit of this act, said applicant may employ to represent him either the attorney of the Massachusetts Medical Society or such other attorney as the applicant may choose and the committee may approve.
3. The Society will pay for the services of such attorney for the defense of the suit against the Fellow only such sum as the chairman of the medical defense committee shall determine to be reasonable.
4. In the event that a Fellow of the Society shall make a request for the benefit provided by this act, the chairman and the secretary of the medical defense committee, acting together, shall have the power to grant the said request or to reject it.
5. The Massachusetts Medical Society shall be under no responsibility for the payment of any sum agreed upon in the settlement of suits, or awarded by the finding or verdict of a court or jury, or for making payments for any purpose whatsoever, except as specified in this act.
6. This act shall take effect upon its approval by the Council and adoption by the Society, and shall apply to all requests for the benefits of this act made after the date of its adoption.

The name "Committee on Medical Defense" has seemed to us better than "Committee on Malpractice Defense," and we advise adopting that name in place of our present designation.

The past year has not been very eventful. Four men have applied for defense and their applications have been granted. Four cases have been disposed of as follows: one case won, two closed with directed verdict for the defendant, and one non-suited.

F. G. Balch, *Chairman.*

APPENDIX NO. 7

Report of the Committee on Permanent Home

The Committee on Permanent Home has nothing new to report at this time. The Building Fund in December, 1936, showed a book value of $57,951.08, with a market value of $51,453. The fund is invested in a diversified list of bonds with a moderate amount of cash accumulating for investment. There has been, as you know, the consideration of two buildings, but up to the present time

no really satisfactory location has been found and the fund is not yet of sufficient size to enable us to purchase exactly what we want. With further accumulations and with the onset of better times we hope to be able to present a more acceptable report.

WILLIAM H. ROBEY, *Chairman.*

APPENDIX NO. 8

COMMITTEES NOMINATED BY THE PRESIDENT TO CONSIDER PETITIONS FOR RESTORATION TO FELLOWSHIP

1. For John H. Andrews, Hyannis
   C. E. Harris, F. A. Binford and J. I. B. Vail.
2. For Oliver E. Bixby, East Lynn
   S. R. Davis, C. L. Hoitt and C. F. Twomey.
3. For Frederick S. Canedy, Wellfleet
   C. J. Bell, H. A. White and F. O. Cass.
4. For Milton M. Cohen, Roxbury
   Maurice Gerstein, Hyman Morrison and Charles Malone.
5. For Ralph W. Dennen, Waltham
   Conrad Bell, O. L. Dascombe and R. H. Wells.
6. For James A. Dumas, Lynn
   N. P. Breed, O. C. Blair and G. H. Kirkpatrick.
7. For Hyman B. Friedman, Brookline
   M. G. Berlin, Cadis Phipps and F. P. McCarthy.
8. For M. M. Gilbert, Lynn
   J. W. Trask, S. R. Davis and N. P. Breed.
9. For Herschel Heinz, New Bedford
   A. H. Sterns, J. M. Salles and L. J. Pobirs.
10. For Nicholas J. King, Roslindale
    J. F. Ford, J. S. H. Leard and Charles Malone.
11. For Joseph Lentine, Boston
    H. F. Newton, Wyman Richardson and L. B. Ellis.
12. For J. B. Toltz, Chelsea
    J. M. Doran, G. B. Fenwick and George Reinherz.
13. For William Worthy, Boston
    A. P. Joslin, E. M. Chapman and H. L. Albright.

APPENDIX NO. 9

REPORT OF THE COMMITTEE ON CANCER

The continued development of the work of the state-aided cancer clinics has done much for both the cancer patient and the physician. Several clinics, notably Worcester and Lynn, celebrated their tenth anniversary this year. The policy of having physicians qualified in special phases of cancer diagnosis and therapy conduct consultation clinics from time to time has met widespread approval.

The program of the Women's Field Army of the American Society for the Control of Cancer has been indorsed by the Council of the Massachusetts Medical Society with the understanding that the objective was to be a membership drive during March to raise funds for the American society, 70 per cent of the sum to be expended locally on a house-to-house canvass to determine the incidence of cancer morbidity. During the spring unfavorable comments from some members of the Society called attention to the deviation of the Women's Field Army in this state from this program.

Although conceived as an activity of the women's clubs, the Women's Field Army was not indorsed by them in this State. The state commander built up an organization which in some instances through enthusiasm and inexperience lost sight of the objectives originally planned and confused the work of the local cancer committees already functioning in many communities.

Through the cordial co-operation of the president and vice-president of the American Society for the Control of Cancer these difficulties are being cleared up, and a united front is presented against this disease. Quite possibly the activities of the Women's Field Army here will be restricted to an annual drive for funds for use by the American Society for the Control of Cancer, an activity in which we can all wish them success. A member of the Women's Field Army on the local cancer committees, rather than an attempt by the Army to build up a parallel or rival organization, would do much to bring about better understanding and progress.

That the efforts of physicians throughout the state are bearing fruit is suggested by the cancer death rate, already showing a slight fall.

We cannot close this report without allusion to Dr. Greenough, long chairman of this Committee. His death is a loss as irreparable as poignant. Keen, resourceful, kind, his activities have brought life and comfort to many cancer sufferers in the past, and development of the principles he laid down will do even more in the future.

SHIELDS WARREN, *Chairman.*

APPENDIX NO. 10

REPORT OF THE COMMITTEE ON POSTGRADUATE INSTRUCTION

On December 18, 1936, the postgraduate extension courses were completed for the academic year 1936-37; this completed the fourth consecutive year of organized postgraduate instruction by the Society. A statistical summary of attendance is appended.

The financial report of the Committee is as follows:

SUMMARY OF FINANCES OF COMMITTEE FOR FISCAL YEAR 1936, AND UP TO JANUARY 15, 1937

Revenue		*Expenses*	
Registration fees	$4,574.00	Faculty, printing, clerical, postage, etc.	$2,935.43
Appropriation	1,000.00	Secretary's salary	1,500.00
		Clerical account to be adjusted	44.00
		Faculty account to be adjusted	46.00
		Possible district expenses	50.00
		1935 overdrawn account	881.68
	$5,574.00		$5,457.11
Less expenses	5,457.11		
Credit balance	$116.89		

At the Council meeting in February, 1937, it was voted to suspend the activities of the Committee until it was found out what plans would be inaugurated by the federal

POSTGRADUATE EXTENSION COURSES

District	1933-34 Active Membership Jan. 1, 1933	1933-34 No. Registered 1933-34	1933-34 Percentage of Membership Enrolled	1934-35 Active Membership Jan. 1, 1934	1934-35 No. Registered 1934-35	1934-35 Percentage of Membership Enrolled	1935-36 Active Membership Jan. 1, 1935	1935-36 No. Registered 1935-36	1935-36 Percentage of Membership Enrolled	1936-37 Active Membership Jan. 1, 1936	1936-37 No. Registered 1936-37	1936-37 Percentage of Membership Enrolled
Barnstable												
Hyannis	37	31	83.7	39	32	82.0	44	29	65.9	48	21	43.7
Berkshire												
Pittsfield	114	74	64.9	118	61	51.6	115	44	38.2	110	41	37.2
Bristol North												
Attleboro	62	19	70.9	66	13	51.5	65	*	24.6	70	*	34.2
Taunton		25			21			16			24	
Bristol South												
Fall River	197	23	37.5	199	22	33.1	208	14	25.9	206	21	32.0
New Bedford		51			44			40			45	
Essex North												
Haverhill	208	64	30.7	198	38	19.1	208	22	10.5	200	*	*
Essex South												
Salem	256	62	24.2	267	53	19.8	267	66	24.7	271	62	22.8
Franklin												
Greenfield	35	29	82.8	34	37	100.0	36	20	55.5	40	29	72.5
Hampden												
Holyoke	285	40	35.0	291	55	35.3	306	26	24.7	297	32	21.5
Springfield		60			48			50			32	
Hampshire												
Northampton	61	40	65.5	63	22	34.9	70	32	45.7	80	29	36.2
Middlesex East												
Melrose	90	32	35.5	102	26	25.4	102	14	13.7	104	13	12.4
Middlesex North												
Lowell	116	59	50.7	117	49	41.8	122	30	24.5	115	37	32.1
Middlesex South												
Cambridge	762	81	10.6	799	65	8.1	842	71	8.4	857	43	5.0
Norfolk												
Boston City Hospital	738	30	10.7	759	*	6.9	767	*	3.7	775	*	1.6
Faulkner Hospital		10			25			*			*	
Norwood		39			28			29			13	
Norfolk South												
Quincy	113	29	25.6	116	35	30.1	108	21	19.4	117	12	10.2
Plymouth												
Brockton	135	53	31.8	127	32	25.1	138	27	19.5	143	20	13.9
Suffolk												
Boston	733	*	*	739	21	2.8	726	*	*	729	*	*
Worcester												
Milford	374	*	*	396	30	15.6	388	24	6.1	393	26	6.6
Worcester		110	26.6		32			*			*	
Worcester North												
Ayer	90	20	45.5	90	16	50.0	96	*	23.9	101	*	23.9
Fitchburg		21			29			23			24	
	4406	1002	22.7	4520	834	18.4	4608	598	12.9	4656	524	11.2

*Course omitted.

and state governments for postgraduate medical instruction in accordance with the provisions of the Social Security Act, which provides certain funds for this purpose. The social security activities for graduate education for doctors are carried out by the United States Public Health Service and the federal Children's Bureau; these two organizations delegate the administration of the postgraduate courses to the health departments of the respective states.

The Executive Committee has had conferences with Dr. Henry D. Chadwick, Massachusetts commissioner of public health, in regard to the proposed postgraduate courses to be given in Massachusetts. He has asked the

Massachusetts Medical Society Committee on Postgraduate Instruction to assist in this undertaking, and has offered to have the Committee organize the faculty and direct the courses in much the same manner as has been done by the Society during the past four years.

It is planned to give a series of eight sessions in approximately seventeen places throughout the State—a total of one hundred and thirty-six sessions.

An estimated budget has been prepared, viz.:

Expense of 136 sessions at $25 per instructor	$3,400.00
Administration and demonstration expense	1,000.00
Transportation for instructors at $.06 per mile on a state-zoned basis	879.84
	$5,279.84

The above budget is to be met by funds provided through the federal agencies as indicated in the Social Security Act, which is now a part of the permanent law of our country. This postgraduate course would be offered without charge to the individual doctor. It is expected that the Massachusetts Medical Society would provide the incidental expenses of printing of programs, clerical assistance and part of administration expense.

The Executive Committee has given this project serious consideration, and recommends that the Council approve the plan of the Committee to co-operate with the state and federal governments in providing postgraduate instruction to the medical profession of the Commonwealth.

It is also recommended that the Committee be continued and directed to co-operate with the governmental agencies in order to carry out the plan of postgraduate instruction as outlined above.

FRANK R. OBER, *Chairman,*
LEROY E. PARKINS, *Secretary.*

APPENDIX NO. 11

REPORT OF THE COMMITTEE ON PHYSICAL THERAPY

Physical therapy is the treatment of diseases or injury by physical methods. These include treatment by the sun, by various types of lamps, by hydrotherapy, by corrective exercises, by massage, and under certain conditions by various forms of apparatus. Its possibilities for practical usefulness to the general practitioner and to the specialist are legion. The recognition of its demonstrable therapeutic advantages is meager and urgently requires emphasis.

For many years the American Medical Association, through its active Council on Physical Therapy and various other channels, has been calling the attention of the medical profession to this still neglected yet powerful armamentarium. There is indubitable need for better education of undergraduate medical students and practicing physicians in relation to both the conditions in which its administration has proved to be an aid to recovery and the methods by which it may be applied most easily and efficiently.

Your Committee requests the co-operation of each member of this Society in the efforts which it is making to solve the problem of undergraduate and postgraduate education in this field. Regardless of the relative value of the various types of application, physical therapy is being employed increasingly and successfully by informed physicians in other states and in other countries. Boston is far behind most centers of medical education in this respect, and we believe, with a few cheering exceptions, that there is even less employment of these valuable therapeutic measures throughout the State as a whole. The members of your Committee consider it their duty and privilege to endeavor to better this state of affairs. Instruction based on fundamental physiologic principles should be provided for undergraduates, not in an attempt to make them specialists in physical therapy but rather to make them more conversant with the general applicability of physical therapy to the treatment of their future patients, and more familiar with the appropriate methods of its administration.

Your Committee is hoping for the appointment of an instructor at each of the three medical schools in Boston, who will be available to all the departments dealing with therapeusis. It is also our ambition to realize the further establishment of physical therapy clinics in teaching hospitals. Without the possibility of demonstration, the didactic instruction of both students and practitioners will be ineffective.

Your Committee is eager to be of service to the district societies by providing talks on the general subject of physical therapy and by staging demonstrations of the different methods of its application at meetings of the various districts. Arrangements for such programs will gladly be made by the chairman of the Committee.

FRANKLIN P. LOWRY, *Chairman.*

APPENDIX NO. 12

REPORT OF THE COMMITTEE ON PUBLIC RELATIONS

Proposed legislation relating to compulsory health insurance has been discussed, and plans for immediate organized opposition to any legislation contrary to principles endorsed by this Society are under consideration. Senator Capper's bill (United States Senate No. 3253) has not as yet been reported out of committee.

The Committee voted that Dr. Hunt be authorized to proceed with the organization of a district medical service council in the Worcester district. This general plan was endorsed by the Council at a prior meeting.

The prepayment hospital plan is proceeding to fruition, a governing body has been elected, and an executive secretary chosen, and plans for inaugurating the system in Metropolitan Boston are under way.

Dr. Landesman's resolution for investigation of Red Cross first-aid stations is referred to Dr. Hunt of this Committee because of his intimate association with Red Cross activities.

E. S. BAGNALL, *Secretary.*

APPENDIX NO. 13

"GENTLEMAN'S AGREEMENT" BETWEEN MEDICAL, HOSPITAL AND INSURANCE INTERESTS

A joint meeting of the representatives of the Hospital Council of the New England Hospital Association, physicians and insurance companies was held at the Medical Library on May 12 at noon. Dr. William G. Curtis presided. Dr. Henry M. Landesman was appointed secretary. The following were present:

Dr. William G. Curtis
Dr. Henry M. Landesman
Dr. John M. Doran, representing the Massachusetts Medical Society
Dr. Henry M. Pollock, of the Massachusetts Memorial Hospitals, Boston
Dr. George A. MacIver, of the Worcester City Hospital, Worcester

Dr. James W. Manary, of the Boston City Hospital, Boston
Dr. Eugene Walker, of the Springfield Hospital, Springfield
William P. Cavanaugh, manager of the Claims Department of the National Bureau of Casualty and Surety Underwriters, New York
John Carr, of the Metropolitan Casualty Company
Frederick J. Reynolds, of the Employers Liability Assurance Corp., Ltd.
Martin L. Hines, of the Travelers Insurance Company
Richard J. Dunn, of the Lumberman's Mutual Casualty Company
James H. Holland, of the Liberty Mutual Insurance Company
Charles E. Wilde, of the Liberty Mutual Insurance Company

The purpose of the meeting was a review by the interests involved of the two "gentleman's agreements," one between the insurance interests and the physicians and the other between the insurance interests and the hospitals, copies of which were printed in a previous issue of the *Journal*. The agreements were considered in detail, and it was generally agreed by all present that the medical and insurance interests should go forward at the earliest possible date and put the agreement into operation. The authorization blanks, copies of which were also printed in a previous issue of the *Journal*, were considered, and with some minor changes were definitely approved.

It was generally agreed that some action should be taken at once in the way of a campaign of education among the doctors, hospitals and insurance companies as to the methods by which the proposed agreements should operate. It was also agreed that the Hospital Council and Hospital Association would make every effort to bring the matter to the attention of hospitals throughout the State, that the medical societies would take action to see that their individual members were advised concerning the agreements, and that the insurance organizations would see to it that individual insurance companies were fully conversant with the agreements and the way in which they should operate. It was understood by all present that questions concerning the agreements would arise, and that some disagreements between the medical and hospital interests and insurance companies would occur, and the general suggestion was that when an individual hospital had a grievance against an individual insurance company, the hospital would at first endeavor to straighten out the grievance with the insurance company, preferably discussing the matter with the claims manager of that company, and that if the hospital and the insurance company could not satisfactorily adjust the matter in controversy, the hospital would then refer the situation to the hospital committee, which would in turn take the matter up with the insurance company committee. The same procedure would prevail in so far as complaints of individual doctors against insurance companies were concerned.

Much discussion was held with regard to the possible effects of the operation of the "gentleman's agreement" with physicians in so far as physicians who were not members of any recognized medical society are concerned. It was recognized that certain of these physicians would not be willing to become a party to any such agreement, but the consensus seemed to be that as the agreement began to operate successfully many of these physicians would readily fall in line with it, and those who would not and whose conduct grossly violated the ethics of the medical profession could be dealt with in a manner to be decided upon at some later date.

All those present evinced a sincere desire to create a spirit of real co-operation between the medical and insurance interests in the handling of automobile personal injury cases within the Commonwealth, and voiced a willingness to do everything possible to make the agreements operate successfully and to the mutual advantage of all parties interested.

WILLIAM G. CURTIS, *Chairman,*
HENRY M. LANDESMAN, *Secretary.*

APPENDIX NO. 14

REPORT OF THE COMMITTEE TO CONSIDER CITY DIRECTORY ADVERTISING

Some months ago, your chairman, as member of a local committee, was approached by a firm of directory publishers to solicit Society members in his district for extended insertion in the list of physicians in the classified section, using a form based on that used in the A. M. A. Directory. Sample lists from the directory in Cleveland were submitted. A short time previously request had been made by a group of younger men for an official list, readily accessible to the public, which might prove their qualifications as against the graduates of the unapproved schools with whom they found themselves in competition. As the directory list seemed to offer possibilities in this direction, permission for solicitation was given. The spread of the plan to other cities and some complaints as to the results made a survey desirable.

The original lists, on which permission for solicitation was given, marked all Society members with an asterisk; the present proposition of the publishers is to restrict this distinction to those taking extended insertion at a fee of $3. A check-up of the sample pages from Cleveland, where the plan is said to have been in operation for five years, showed only about 20 per cent so listed; this at once threw doubt on the value of the scheme for our purposes and indicated little enthusiasm for the plan. Among others, the chairman of the A. M. A.'s Judicial Council (corresponding to our Committee on Ethics and Discipline), a resident of Cleveland, was not so listed.

The proposed form of listing also included specialties, and the question of criteria immediately arose. The Cleveland list showed odd classifications, such as "varicose veins and ulcers," "cardiac ailments," "physician and surgeon," "x-ray and internal diseases" and "obstetrics and female diseases."

These lists were evidently made up on the statement of the individual doctor. We considered listing only those who had passed the national boards in their specialties. But all the specialty boards are not yet functioning. We learned further that, complaints having been made regarding the specialist listings in the yellow section of telephone directories, the New England Telephone and Telegraph Company had agreed to suspend this feature.

The situation at present is confused; we feel that no further sanction should be given the idea. But in justice to the publishers and to members who may favor such a listing, your Committee is willing to continue in office if desired and watch further developments such as might be entailed by the adoption of a general standard for specialists. For the original purpose, we rather doubt the value of directory listing. In one district, the suggestion has been made of an inexpensive booklet, like those used by Masonic lodges, to be furnished at cost to members for general distribution.

PHILIP H. COOK, *Chairman,*
J. FORREST BURNHAM,
ROBERT L. DENORMANDIE.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 23301

PRESENTATION OF CASE

A 54-year-old Irish painter was admitted complaining of painful ulceration of the left foot.

About a year before entry the patient first noted a crawling sensation on the inner aspect of the left calf. This sensation occurred intermittently and appeared only at night. It was never associated with pain but frequently kept the patient awake. Eight months before entry he noted that the left leg felt cold and there was a deep-seated, dull, aching pain, at first limited to the calf but later involving the entire extremity below the knee. The pain was constant but was aggravated by walking and caused the patient to walk with a limp. On occasion it was necessary for him to stop walking and shake his leg because it felt "as though there was no circulation in it." There was no evident discoloration. Four months before coming to the hospital the patient was awakened by pain in the left foot. Stepping on the extremity caused extreme pain which, however, subsided after the foot had been on the floor for about 5 minutes. At this time the foot became discolored and a scratch, which had been present on the lower leg without healing for several months, gradually became larger until it surrounded the entire extremity and involved an area about 12 cm. above the ankle. The ulcer was extremely sensitive and painful, and the foot exhibited intermittent swelling. Shortly before entry an area of bluish discoloration appeared on the dorsum of the foot. There was considerable pain associated with a sensation of numbness, and the movement of the toes was impaired. For about 5 months there had been intermittent, deep-seated, aching pain in the right leg, which on occasion became swollen.

Four months before entry a physician elicited a story of a penile sore 31 years previously. A blood test was done, after which the patient received intravenous antisyphilitic therapy and iodides by mouth. Three weeks prior to admission an itchy, dry, red rash suddenly appeared upon the patient's body and extremities. Intravenous injections and iodides were discontinued, and the rash became scaly but improved slightly.

Two years before entry he suffered for a short time from severe palpitation. A physician stated that he had "nothing to worry about," and suggested that he "take it easy."

Physical examination showed a moderately pale, well-developed and nourished man suffering from considerable pain in the leg. The pupils reacted sluggishly to light. There was a dry, scaling erythematous eruption distributed diffusely over all of the extremities and in a patchy fashion on the trunk. There was a deep, ragged, foul-smelling ulcer encircling the entire left lower leg for a distance of about 15 to 20 cm. above the ankle. The edges of the ulcer were irregular and granular in appearance, and the base was necrotic and extended to the underlying bone. There was pitting edema of both feet, more marked on the left side. The chest was resonant, and a few fine rales were audible at the lung bases. The left border of cardiac dulness extended 13 cm. from the midsternal line, and the supracardiac dulness was broadened, although no measurement was recorded. The apex impulse was in the sixth interspace and was variable in intensity. The heart sounds were grossly irregular in rhythm and variable in intensity. A systolic murmur, unaccompanied by a thrill, was heard at the base of the heart and was transmitted into the neck. The blood pressure was 155/80. The remainder of the examination was negative. The reflexes were normal. No note was made of the character of the peripheral vessels.

The temperature was 102°F.; the pulse 110. The respirations were 30.

Examination of the urine was negative. The blood showed a red cell count of 4,360,000, with a hemoglobin of 65 per cent. The white cell count was 8,650, with 82 per cent polymorphonuclears. A Hinton test on the blood was positive, a Wassermann was negative.

On the second hospital day a guillotine amputation just below the left knee was performed. Postoperatively the patient's temperature returned to normal, where it remained with only an occasional rise to 99.8°F. throughout the period of hospitalization. His general condition improved considerably despite the appearance of a small decubitus ulcer on the right hip. Digitalis was administered, and the auricular fibrillation ceased. On the tenth postoperative day the patient was permitted to be up in a wheel chair. He felt comfortable until evening, when he complained of transient, vague, substernal discomfort. This subsequently disappeared, and the patient felt quite well. Shortly afterward he was found dead in bed.

Differential Diagnosis

Dr. Henry H. Faxon: I am somewhat embarrassed to discuss the medical aspects of this case as it seems to me to require specific medical knowledge concerning which the surgeon is apt to have become somewhat hazy. So I will confine my discussion to the surgical aspects of the leg and will assume, more or less arbitrarily, that this 54-year-old painter had cardiac damage and syphilitic aortitis, and that he had circulatory trouble in the extremities with superimposed infection that precipitated amputation. I do not believe that there is any question of lead poisoning, or that the fact that he was a painter played any part in his disease. I assume that the rash was from the iodides that had been administered, and that his sudden death was more probably from coronary disease than from embolus because of the interval of complete freedom from symptoms between the first episode of substernal discomfort and the later finding of sudden death.

So far as the local condition of the extremity is concerned, there are a number of ways of approaching the problem. Could the extensive ulcer that he had be purely local, and not on a circulatory basis? In other words, Could it be a skin malignancy, tuberculosis, or some form of symbiotic ulcer or unusual infection? The man very definitely gives an antecedent story of some 6 months of difficulty with the extremity before the lesion appeared, which would be against these diagnoses. From a circulatory point of view we can ask the question whether his extremity and the ulceration were due primarily to circulatory difficulty. There is no note of pulsations, and there is nothing to suggest any venous abnormality. Vasomotor spasm and Raynaud's disease can be excluded by the story of the patient's advanced age. Buerger's disease, or thromboangiitis obliterans, is seen ordinarily in the younger age group. It almost invariably affects the more distal portion of the limb rather than the higher level that this man complained of, and I can see little to recommend it as a diagnosis.

An aneurysm in the popliteal space would be detected on physical examination, and some note would have been made of it. The story is too long to be attributed to an embolic origin. Straight uncomplicated arteriosclerosis as we see it in patients of advanced years does not as a rule give the type of story brought out in this case. In the arteriosclerotic group intermittent claudication is almost invariably the first symptom of discomfort that the patient experiences. Pain of the type mentioned in this history would be a most unusual finding, for although the arteriosclerotics often are awakened with a tingling sensation in their feet in the early hours of the morning, this is rarely an initial complaint as in this case. I think definitely that this patient had a certain amount of peripheral sclerosis and that the circulation in the limb was somewhat impaired. The fact that walking aggravated his pain bears out the impression that with increased activity there was a certain amount of anemia of the tissues. Had his lesion been purely on an arteriosclerotic basis, however, the patient would almost certainly have observed that his foot became cyanotic on dependency.

The fact that he was awakened at night by pain in the left foot might suggest an embolic affair, but it would be rare to have pain of embolic origin localized to the foot and relieved rather than aggravated by dependency.

The significant thing about the ulcer is its location. It was 12 cm. above the ankle. It had become progressively worse since its onset. With almost all of the lesions attributable directly to circulatory changes in the extremity, the first areas of ulceration or gangrene are on the distal portion and involve perhaps the anterior portion of the foot, or one or more toes; whereas this man for a period of 4 months after the onset of the ulceration of the lower leg had noted little change in the foot itself. I wondered if the pain he was having in his legs might possibly be that of tabes and the ulcer a trophic one on a neurologic background. However, the facts that his reflexes were reported as normal and that the area of ulceration was hypersensitive rather than anesthetic exclude this possibility.

The next point is the fact that both extremities were apparently involved. So I do not think that the trouble in this case was primarily confined to one leg, but do believe that a certain amount of anemia of the leg was a contributing factor in the difficulties he ran into. Looking at it another way, Could most of the trouble be due to his general systemic condition? I am more inclined to think that rather than the usual straightforward arteriosclerotic type of case this man's primary trouble was due to a cardiac condition and secondarily to changes that occurred in his leg.

In summary, I believe that this man had syphilitic aortitis with cardiac enlargement and decompensation, and that the changes in his vessels were of secondary importance. Undoubtedly at 54 he would show a certain amount of arteriosclerosis, but syphilitic aortitis plus cardiac decompensation was more probably the basis of peripheral anemia than was the local condition itself. His death was probably the result of coronary disease.

A Physician: What about the thickened area shown by x-ray at the middle third of the tibia?

Dr. Faxon: The ulcer goes down to the bone,

and I think any ulcer that is that deep will inevitably produce a good deal of associated periostitis.

A Physician: We know that plain syphilitic ulcer of the skin will give a picture like this.

Dr. Faxon: I am at a loss to say what was the exact cause of the ulceration but believe that it was due more to his general systemic disease with syphilis as one of the factors than to local circulatory causes with resulting local anemia.

A Physician: You do not think that the vessels in the leg showed sclerosis?

Dr. Faxon: I do not think that they will show more than the degree of sclerosis that might be expected in a man of his age.

Dr. Wyman Richardson: There is one other point here, probably put in to make it a little more difficult. Auricular fibrillation is said to have stopped just before death. That would be a good argument for the appearance of an embolus about that time.

A Physician: He had a septic amputation stump which might have been a focus for an embolus.

Dr. Faxon: The reason I said coronary disease, somewhat reluctantly, rather than embolus was that after the episode of substernal pain he had a period of apparently complete recovery before he was subsequently found dead. However, embolus is a possibility.

One other word in regard to syphilitic lesions of the leg. Although we have seen a great many ulcers in the Circulatory Clinic we have seen very few that were syphilitic and these, when present, have as a rule responded well to specific treatment.

Clinical Diagnoses

Arteriosclerotic heart disease.
Gangrene of the leg.
Pulmonary embolus?
Coronary thrombosis?

Dr. Henry H. Faxon's Diagnoses

Syphilis with aortitis and cardiac decompensation.
Ulceration of the left leg (noncirculatory in origin).
Peripheral arteriosclerosis.
Coronary thrombosis.

Anatomic Diagnoses

Surgical specimen:
- Arteriosclerosis, diffuse, without thrombosis.
- Infectious ulcer, probably symbiotic, of lower leg.

Postmortem:
- Arteriosclerosis, marked, aortic, coronary and peripheral.
- Coronary thrombosis, old, circumflex branch of left, main artery, right.
- Myocardial infarction, fibrotic.
- Amputation of left lower leg.
- Decubitus ulcers, multiple.

Pathological Discussion

Dr. Mallory: A few weeks ago I had occasion to look over, with the aid of Dr. Gall and Dr. Derrick, a series of 1,000 successive autopsies, comparing the clinical diagnosis with the postmortem diagnosis. That was done in preparation for an informal talk to the Harvard Medical School class of 1912, which has just had its twenty-fifth reunion. The point that interested me was that in 1912 Dr. Richard C. Cabot published a similar series of cases based on a comparison of clinical and anatomic diagnoses in 1,000 autopsies, and I thought it would be amusing to compare our present record with that which Dr. Cabot reported 25 years ago. The results were rather interesting and, I confess, surprising. The percentage of correct diagnoses was practically identical, with 3 or 4 per cent in favor of the 1912 series. That was particularly true in regard to the subject of pulmonary embolus. Probably the most frequently missed diagnosis in this hospital is pulmonary embolus, and it is missed both ways. It is diagnosed when not present, and missed when present.

Having done my best to worry Dr. Faxon, I will now say that this patient did not have a pulmonary embolus but died undoubtedly of coronary disease. All branches of the coronary arteries were tremendously narrowed. Several were completely occluded with old thrombi. The myocardium showed many patches of fibrosis, but no fresh infarction. Neither the aorta nor all the rest of the body showed anything on which we could make a diagnosis of syphilis. He had a marked degree of arteriosclerosis, but no aortitis. The vessels of his extremities were rather markedly sclerotic, but I think Dr. Faxon was correct in his analysis of the condition in the amputated left leg. Although the vessels were markedly narrowed they were nowhere completely occluded, so I do not think that this very extensive ulceration can be blamed upon arterial occlusion. We failed to explain it adequately ourselves. There was nothing on which we could make a diagnosis of gumma, and I do not believe that it was syphilitic, but on the other hand I do not believe that we ruled out the possibility of symbiotic ulcer. The general character of the ulcer, which was very deep and seemed almost like a surgical dissection exposing the tendons and periosteum, was very much like that of a symbiotic ulcer. I agree with Dr. Faxon that several separate factors probably contributed to the production of the lesion.

Dr. Richardson: What is your feeling in regard to the relation of exposure to lead and arteriosclerosis?

DR. MALLORY: It is generally considered that there is a relation; practically speaking, we have seen very little evidence of it uncovered here, and I do not feel that I have any personal experience to bring to bear on it.

DR. RICHARDSON: My impression clinically is that we do see that relation.

CASE 23302

PRESENTATION OF CASE

A 35-year-old Canadian truck driver was admitted complaining of bloody diarrhea.

The patient had been entirely well until 3 months before entry, at which time, without premonitory symptoms, he began to have frequent watery and bloody bowel movements containing strings of mucus. Diarrheic movements were preceded by cramplike pains in the lower abdomen associated with an urgent desire to defecate. Following each bowel movement there was transient subsidence of cramps, but they recurred within ½ to 1 hour. In all, there were twelve to fifteen movements daily, but none at night. Despite the fact that his appetite was unimpaired he lost about 15 pounds in weight during the 3 months of his illness. Three weeks before admission he consulted a physician who prescribed a diet. Following this the bowel movements diminished in frequency, but rectal bleeding, which had become quite profuse, remained unchanged. There had been no fever, nausea or vomiting. One week prior to entry he developed a sore throat and head cold. This, associated with his diarrhea, caused him to feel very weak, and he decided to enter a hospital.

The patient had been married 10 years, during which time his wife had had three normal deliveries and three spontaneous miscarriages. His past history was noncontributory.

Physical examination showed a well-developed, fairly well-nourished young man in no acute discomfort. The patient was thin and appeared to have lost a moderate amount of weight. The pharynx was slightly reddened. The lungs were clear. The heart was normal. The blood pressure was 115/80. The abdomen was thin, and there was slight generalized voluntary spasm. Loops of small intestine were palpable, but there were no abnormal masses. A proctoscope passed for a distance of 15 cm. elicited slight spasm. Many raw, bleeding, shallow ulcerations were seen, and there was considerable pus and mucus. The mucosa was granular, edematous and reddened, with loss of normal sheen. There were occasional scarred areas. Smears from the ulcer bases showed many red blood cells but no amebae.

The temperature, pulse and respirations were normal.

Examination of the urine was negative. The blood showed a red cell count of 4,200,000, with 75 per cent hemoglobin. The white cells numbered 6,850, 74 per cent polymorphonuclears. Repeated stool examinations showed positive reactions to the guaiac test. A blood Hinton test was negative. The serum chlorides were 102 milliequivalents. The nonprotein nitrogen of the blood was 20 mg. per cent. The serum protein was 6.4 gm. per cent, the serum calcium 10.7 mg. per cent, and the phosphorus 3.7 mg. per cent. A Widal test was negative, as were agglutination reactions for paratyphoid and undulant fevers. Stool cultures were negative for pathogenic organisms. A Frei test was negative.

X-ray of the chest showed a primary Ghon's tubercle in the left lower lung field, with corresponding calcified hilar glands. The remainder of the chest film was negative. A barium enema passed to the ileocecal valve. The sigmoid was somewhat smoother than normal at its juncture with the descending colon. It was fairly flexible, however, and after defecation no definite changes in the mucosa were demonstrated.

During the succeeding month the patient showed gradual improvement upon a low residue, high caloric, high vitamin diet. He received eleven 1 cc. injections of emetin hydrochloride, and the bowel movements diminished to one or two a day, although there was an occasional passage of a small amount of pure blood. The temperature remained normal except for an occasional rise to 100°F., and the patient gained 5 pounds. On the thirtieth hospital day he again complained of vague abdominal discomfort, and the bowel movements increased to eleven per day. Subsequently, lower abdominal pain increased in amount, and there was generalized rebound tenderness. Peristaltic waves were audible. The temperature rose to 104°F. and the white blood cells to 16,000. A plain film of the abdomen showed no free fluid. The patient was placed in Fowler's position and given sedatives and fluids parenterally. Thereafter the temperature fluctuated irregularly between 101 and 104° F., and a firm, soggy, tender mass became palpable in the patient's lower quadrant. Rectal examination was negative. His condition remained essentially unchanged until the forty-fifth hospital day, when he had a sudden severe pain in the abdomen requiring two injections of morphine for relief. During the succeeding 24 hours there were no bowel movements, the abdomen became distended and markedly tender, and no peristaltic sounds were heard. His condition became rapidly worse, and he died on the forty-seventh hospital day.

Differential Diagnosis

Dr. William B. Breed: This is a very acute and rapidly progressive process for what it seems to be, that is, some sort of colitis. Apparently 4 months before his death he was in good health. Then the outstanding symptom, diarrhea, began, with an unusual amount of blood. Many of the stools instead of being loose and watery were made up of pure blood. There is one significant statement in the history, unconfirmed by any further note—he had no bowel movements at night. We are accustomed to infer when a person with diarrhea has no bowel movements at night that the disease is not of an organic nature. However, we know that this is organic diarrhea, so to speak, because of the persistence of blood, and we also know that the patient died of a disease centering in his colon; so that we cannot place too much emphasis on such a statement in this case. There are not many diseases that will produce this picture, and I think it is just as well before we go any farther for us to put down the various pathological possibilities, and then with the record before us rule out as many as we are able. Without listing them in order of their probability, we should consider: (1) amebic dysentery, (2) tuberculous enteritis, (3) ulcerative colitis and (4) bacillary dysentery.

There is very little in his past history to help us. The first objective evidence is that he was thin, and showed a moderate amount of loss of weight, but he was apparently not uncomfortable and had no fever. Physical examination showed generalized voluntary spasm and no masses in the abdomen. The proctoscopic examination makes a diagnosis of colitis perfectly definite; whether this is ulcerative colitis or an amebic infection, we cannot tell definitely, because the best of proctologists will sometimes err in this distinction. It has happened a number of times that a definite diagnosis of ulcerative colitis has been made by proctoscope and the patient has been cured by antiamebic treatment. It does not seem to be tuberculous. Bacillary dysentery does not give this picture in the rectum and sigmoid.

The laboratory data are of no value in coming to a conclusion. All the chemical values were within normal limits. The Widal and other agglutination tests were negative. The Frei test was done, I take it, because of the note that there were seen occasional scarred areas by proctoscope, which might suggest a beginning stricture due to lymphogranuloma inguinale. In that disease one usually finds enlarged inguinal lymph nodes and a process in the rectum which eventually produces a stricture.

Let us look at the x-rays. The chest is entirely negative except for a calcified tubercle, the so-called Ghon tubercle, with the associated hilum calcification that indicates a tuberculous infection during childhood, and is of no significance. There is no acute pulmonary process, which lends very strong evidence to the conclusion that this is not tuberculous enteritis. The colon by x-ray is entirely negative, and one can definitely feel sure that this is not chronic ulcerative colitis, because if it were the x-ray would show a small contracted colon, with loss of haustral markings, and so forth. This is a normal colon by x-ray, before and after evacuation. I cannot pick out the smooth sigmoid at its juncture with the descending colon. We have to conclude that the x-ray of the colon is entirely negative and that we can rule out chronic ulcerative colitis, but not acute ulcerative colitis.

Apparently they followed the routine that we have in this hospital of giving a course of antiamebic treatment. To be sure he did improve somewhat, but I should infer that if he had improved enough on this treatment to make them believe it was an amebic infection they would have continued the treatment. So from the record I think we can confidently rule out amebic dysentery.

We have ruled out tuberculosis, chronic ulcerative colitis, amebic dysentery and bacillary dysentery, which leaves us with an acute ulcerative colitis. This fits in with his story because he lived only 4 months after the onset of his disease. It is possible to have acute ulcerative colitis limited in its location, that is, largely in the rectum; but this colon may be acutely affected even though the barium enema does not tell us anything. My conclusion, therefore, is that this man had a rapidly progressive acute ulcerative colitis, and I should predict that we would find the whole colon seriously affected. I think that he had a perforation at the end, and that he died of peritonitis. It is surprising how rapidly progressive some of these cases can be. It is not at all unusual for ulcerative colitis to be as acute as this. When we talk of ulcerative colitis we must not fall into the habit of always calling it "chronic."

A Physician: It is not so acute. He has had it for 4½ months.

Dr. Breed: It is, compared with the chronic type. Chronic ulcerative colitis often goes on for years. I think that in relation to chronic ulcerative colitis this is acute, and after all it is a relative matter. Whether we call it acute or chronic, it is a rapidly progressing affair.

A Physician: Was emetin all that was given in the way of treatment?

Dr. Breed: According to the record that is all he had for some time.

Dr. Benjamin Castleman: The stools were cultured and no amebae found. Besides emetin he

was given a high vitamin diet, bismuth subcarbonate and morphia.

Dr. Breed: They gave the emetin at entrance; I believe that is routine here in these cases.

A Physician: How about the possibility of chemical poisoning?

Dr. Breed: There is nothing about it in the record, and that is all we have to go on.

Dr. Castleman: He was seen by a surgeon who was not sure that he had perforation. He could not rule out an acute exacerbation of the colitis with peritonitis on top of that. He decided not to operate because the patient was too sick.

Clinical Diagnoses

Chronic ulcerative colitis.
Peritonitis.

Dr. William B. Breed's Diagnoses

Acute ulcerative colitis.
Perforation peritonitis.

Anatomic Diagnoses

Chronic ulcerative colitis (acute phase) with perforation.
Peritonitis, generalized, acute.
Fatty vacuolization of the liver, marked.
Pulmonary alveolar hemorrhage.
Pleuritis, chronic fibrous, left.
Hydrothorax.
Arteriosclerosis, minimal, aortic and coronary.

Pathological Discussion

Dr. Castleman: At autopsy the abdominal cavity contained about a liter of foul-smelling fecal material and showed diffuse generalized peritonitis. We found a perforation at the rectosigmoid about 0.5 cm. in diameter. As is common, the ulcerative colitis was more severe in the terminal part of the large bowel. The whole colon was very friable and soft, and just by handling it it perforated. As we went higher up toward the cecum there were fewer ulcerations but it was still involved up to the ileocecal valve. In many areas there were pseudopolyps, that is, the remaining mucosa between ulcerations projecting into the lumen and simulating true polyps. However, no true adenomatous polyps such as those which develop in the more chronic type of the disease were present. Microscopically the inflammatory exudate was composed of polymorphonuclear leukocytes, lymphocytes and mononuclear cells. The ulcerations had extended in many places deep into the muscularis, and in the region of the perforation there was an acute fibrinous exudate on the serosa.

Dr. Breed: There was nothing in the small intestine?

Dr. Castleman: No. It is quite rare for the disease to extend into the small intestine.

He had bronchopneumonia and a pleuritis due to old tuberculosis. The liver was very soft and fatty.

The New England
Journal of Medicine
Formerly the
Boston Medical and Surgical Journal
Established in 1828

Owned by the Massachusetts Medical Society and Published Under the Jurisdiction of the Committee on Publications

Official Organ of
The Massachusetts Medical Society
The New Hampshire Medical Society
The Vermont State Medical Society

Editorial Board

George G. Smith, M.D.
William B. Breed, M.D.
George R. Minot, M.D.
Frank H. Lahey, M.D.
Shields Warren, M.D.
George L. Tobey, Jr., M.D.
C. Guy Lane, M.D.
William A. Rogers, M.D.
Dwight O'Hara, M.D.
John P. Sutherland, M.D.
Stephen Rushmore, M.D.
Hans Zinsser, M.D.
Benjamin White, Ph.D.
Henry R. Viets, M.D.
Robert M. Green, M.D.
Charles C. Lund, M.D.
John F. Fulton, M.D.
A. Warren Stearns, M.D

Associate Editors

Joseph Garland, M.D.
Thomas H. Lanman, M.D.
Donald Munro, M.D.

Walter P. Bowers, M.D., Editor Emeritus
Robert N. Nye, M.D., Managing Editor
Clara D. Davies, Assistant Editor

Subscription Terms: $6.00 per year in advance, postage paid, for the United States; Canada, $7.04 per year; $8.52 per year for all foreign countries belonging to the Postal Union.

Material for early publication should be received not later than noon on Saturday.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to the New England Journal of Medicine, 8 Fenway, Boston, Mass.

SIR SQUIRE SPRIGGE

In the death of Sir Squire Sprigge, June 17, 1937, medicine loses one of its outstanding figures. For some thirty years he was editor of the *Lancet,* a journal which takes the leading place in the medical literature of the world. All will agree that no journal is better edited than the *Lancet,* although in recent years others have come nearly up to the standard set by this famous medical periodical.

Sir Squire Sprigge was born in Norfolk, England, the son of a country doctor and small landowner. He first went to Cambridge University and then to St. George's Hospital, London, from which he graduated in 1887. While there he was a popular, energetic student, noted for his charm of manner and wit. From an early age he had been interested in literature, particularly the short story, and his first efforts after leaving the hospital were in this field. He became the secretary of the Society of Authors, founded about that time by Sir Walter Besant. It was with Besant that Sprigge first visited America, coming to the Chicago World's Fair in 1893. There his striking personality was soon noted, and he was freely caricatured in the American press. It was in Chicago, moreover, that he received a telegram offering him a position in the editorial room of the *Lancet.* He returned at once to London and took up this position, to which he devoted his entire time until within a few days of his death in his seventy-seventh year. In 1908, after the death of T. H. Wakley, the grandson of the founder of the *Lancet,* Sprigge was appointed editor.

He was particularly fortunate in the men with whom he was associated in his early days at the *Lancet* office—Dr. H. P. Cholmeley, a fine scholar, and Dr. N. Gerard Horner, who later became the editor of the *British Medical Journal.* During the early years of the World War, when his staff was greatly depleted, Sprigge edited the journal almost single-handed. Later he was assisted by Dr. Egbert Morland. The *Lancet* grew and widened its influence under Sprigge's editorship. He was recognized as a man of outstanding ability by both his colleagues and the general public. In 1921 he received a knighthood and was elected a fellow of the Royal College of Surgeons.

As the editor of the *Lancet* Sprigge made a second visit to America in 1928. While here he attended a meeting of the American College of Surgeons in Boston and delivered a striking address, entitled, "Grand Curiosity, as Exemplified in the Life of John Hunter." This was published in the *Lancet,* October 13, 1928, and also in *Surgery, Gynecology and Obstetrics* for December of the same year. At the time of his visit to Boston, he called upon the then editor of the *New England Journal of Medicine.* A few months prior to this, the *Journal* had changed its name from the *Boston Medical and Surgical Journal,* after a continuous existence of one hundred years. Sprigge, naturally, was much interested in the anniversary of the *Journal,* with its unique record in this country and exceeded only by one other medical journal in the world, the *Lancet.*

Sprigge was a versatile man, of wide interests; he painted in water colors with more than ordinary talent and was exceptionally good at playing games. He wrote a number of books of short stories, issued when he was a young man; some of them were later republished. In addition, he issued a volume of essays, "Physic and Fiction," wrote a life of Thomas Wakley, the famous founder of the

Lancet, and published numerous papers in the journal itself, most of them appearing anonymously.

Services in his memory were held in the church of St. Martin-in-the-Fields, Trafalgar Square, near the former office of the *Lancet* in Bedford Street, Strand, on June 22, at which Viscount Dawson of Penn, president of the Royal College of Physicians of London, expressed his admiration for Sprigge and his work. In the *Lancet* for June 26 and July 3, there are many eloquent tributes to the late editor of this splendid publication. A striking portrait is reproduced in the former issue. His death called forth tributes from almost every branch of medicine and also the world of literature. As Lord Dawson truly said in his address at the memorial services, Sprigge "was a leader by the force of example, the power to persuade and encourage others and to make his workers feel themselves to be friends engaged in a common enterprise; and his kindness had a sureness of touch which brought all men to him."

HOLLYWOOD SURGERY

We are in receipt of a communication from a fellow of the Massachusetts Medical Society calling attention, in a laudably condemnatory fashion, to a recent ambulance dash that was featured conspicuously in the daily press. In this particular instance, a patient with appendicitis was rushed under police escort, through heavy Sunday traffic, thirty miles from Franklin to a Boston hospital. The distance was covered in forty minutes. The patient, in all probability, was in less immediate danger than dozens of innocent travelers whose lives were placed in jeopardy by such a spectacular escapade. The fact seems to have been overlooked that much nearer to Franklin than Boston could have been found hospitals and surgeons thoroughly capable of handling the situation in an adequate fashion.

We do not know the inward reaction of the police who are asked to chaperon these melodramatic adventures. Perhaps, like riders of the pony express, they like the excitement; more probably they feel that they are doing their dangerous bit in helping to save human life; quite possibly they doubt the wisdom or the necessity of many of these mad dashes, but realize that they are not qualified to judge each case on its individual merits. Our correspondent is inclined to believe that cool thinking might easily have determined better ways of handling the situation than rushing around, clanging ambulance bells and cutting corners on two wheels, the while the patient is suffering the results of such handling rather than being assisted toward safety and security.

It would be absurd to question the fact that occasionally emergencies arise where speed in getting a patient to the hospital is of prime importance, and speed must be employed so far as it is consistent with the public safety. Such cases can easily and honestly be explained to the authorities who can then arrange the matter of transportation in the most expeditious and at the same time the safest manner. These instances, however, must be comparatively few and far between. Our correspondent suggests that a consultation between physicians might frequently be possible and always appropriate before a spectacular course of action is decided upon.

IMPROVED CARE OF DIABETICS

The New York Diabetes Association is an organization developed under the aegis of the New York Tuberculosis and Health Association. Now in its third year of growth, it has three main objectives: educational activities; services such as organization of a summer camp attended by 31 diabetic children last year and identification cards for diabetics taking insulin; and general activities such as a survey of the various diabetic clinics in New York City, the study of care of the feet, and a survey of hospital facilities for diabetic patients.

This association, endeavoring to promote interest in and better care of such an important disease, is well deserving of the support it has received. The summer camp for diabetic children is modeled along the lines of those established some time ago by Doctor Elliott P. Joslin, which have proved their worth many times over in giving to diabetic children the advantages of camp life plus education in the care of their disease and means of compensating for its presence. This year the five diabetic camps in New England will care for a total of about 190 children, coming from many hospitals and diabetic clinics, some even from the Ohio valley.

These camps are pleasantly situated at Oxford, on the Clara Barton Homestead; at the Preventorium at Mattapan and the camp at Winthrop of the Boston Tuberculosis Association; at Lake Winnepesaukee, New Hampshire; and at Roxbury, Vermont.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY

M. Fletcher Eades, M.D., *Secretary*,
19 Bay State Road,
Boston, Mass.

Case History No. 30. Miscarriage at Five and One-Half Months

Mrs. N. a white woman, aged 25 years, was first seen at her home on May 27. Her last menstrual period had occurred January 26. Except for slight nausea and occasional morning vomiting the first 3 months, the patient had felt very well. On May 20, slight staining was observed which continued off and on for 1 week. Free flowing then started with a few cramps in the lower abdomen, which induced the patient to go to bed and send for a physician.

There was no family history of tuberculosis, diabetes, malignancy or hemorrhagic diseases. The patient's health had always been good except for childhood diseases, and lobar pneumonia 4 years previously. The menses began at 13 and had always been regular, painless and normal in amount. There had been no previous pregnancies and no surgical operations.

Physical examination revealed a well-developed, well-nourished but somewhat apprehensive young woman with good color of the skin and mucous membranes. The temperature was 98.4°F. The tongue and throat appeared normal, and the thyroid was not enlarged. Chest expansion was good, the breath sounds were normal, and there were no rales.

The size of the heart was normal, and there were no murmurs. The sounds were regular and of good quality, with a rate of 84 per minute. The blood pressure was 118 systolic, 74 diastolic. The abdomen was relaxed, and there was no spasm or tenderness. A symmetrical mass, apparently the uterus, could be palpated four fingerbreadths above the symphysis pubis. The patient had soaked three pads in the past 4 hours and was having slight cramplike pains in the lower abdomen. One-quarter grain of morphia was administered, after which the flow diminished; the cramps ceased, and the following day, except for slight staining, there were no symptoms.

Three days later she was permitted to get out of bed. Automobile riding, coitus and strong cathartics were forbidden, and she was advised to return to bed if a showing of blood recurred.

A series of selected case histories by members of the section will be published weekly.

Comments and questions by subscribers are solicited and will be discussed by members of the section.

There were six similar experiences after the first attack, but each subsided under rest in bed and sedatives. The fundus continued to enlarge, and quickening was noticed June 5. Inspection of the cervix between flowing spells revealed no erosion, polyp or other neoplasm.

On July 12 a profuse flow started and continued with the passage of two large blood clots. The patient was transferred to the hospital. After the usual preparation a vaginal examination was made, and the cervix was found to be one finger dilated and one-half effaced. The fundus was two fingerbreadths above the umbilicus. The flowing had not diminished, and a diagnosis of inevitable miscarriage was made. The bladder was emptied, the blood clot removed from the vagina and a Number 4 Voorhees bag passed through the cervix and filled. A firm Spanish windlass was then applied to the abdomen. The patient's pulse was 118 and of fair quality, and the blood pressure 104 systolic, 56 diastolic. A donor was secured, but as there was no further bleeding and the patient's condition improved after the administration of 500 cc. of 10 per cent glucose by vein, transfusion was not done. Morphine sulfate, ¼ gr., was given and a ½ lb. weight was attached to the bag.

Four hours later the bag was expelled, and a few minutes later a male fetus was delivered spontaneously. It weighed 1 lb. 14 oz. and lived 3½ hours. The small placenta showed a brown, depressed area containing old blood clot. There was very little bleeding during or following the third stage, and the uterus contracted well. The following day the temperature was 99.6° F., the pulse 96 and respiration 20. A blood count showed 3,100,000 red cells, with 60 per cent hemoglobin (Sahli). Ferrous sulfate, 6 gr. four times a day, was prescribed, and a high protein diet was given. The patient made an excellent recovery and was discharged on the twelfth day.

CORRESPONDENCE

SHOCK THERAPY

A recent editorial in this *Journal* (**216**:944 [May 16] 1937) describes the insulin shock treatment of schizophrenia. One must agree with the writer that anything which may ameliorate this scourge is to be hailed as an important advance. In all probability even such severe treatment as repeated insulin shocks may occasionally be justified when the prognosis is bad—and it frequently is extremely bad in schizophrenia. On the other hand one cannot wholeheartedly share the optimism of the proponents of this method of treatment. In the first place shock therapy is not new. It has long been known that any situation which brings a schizophrenic patient near to death may rid him temporarily of his mental symptoms. Thus many major operations (if really major enough) for "the removal of foci of infection" have caused remissions in mental symptoms—sometimes fairly long remissions and frequently short ones. Witness the colectomies prac-

ticed some fifteen years ago on many inmates of mental hospitals.[1] Moreover, it has long been known to psychiatrists that a severe infectious illness occurring in a schizophrenic patient, or a disease requiring operation, may cause the mental symptoms to disappear for a time. Giving these patients convulsions with "metrazol"[2] has had a similar effect, and periods of hyperpnea induced by breathing carbon dioxide and oxygen have brought about brief lucid intervals.[3] The only factor common to these different procedures is a severe metabolic change amounting to shock. It can be reasonably argued that the effective mechanism common to these different shocks may be psychologic — the fact that the schizophrenic patient is faced with a great reality—the fear of death. This state of terror is first induced and then relieved by a physician, and a new emotional situation is brought about.

Bearing these possibilities in mind, one should read carefully two recent contributions presented at Atlantic City this June which give evidence that insulin shock may cause cerebral lesions. At the meeting of the American Neurological Association, Keyes, Freed and Riggs reported a case where insulin treatment for schizophrenia caused a prolonged convulsion, during which hemorrhage took place into the subarachnoid space, accompanied by hemiparesis and prolonged stupor. They report finding petechial hemorrhages in the brains of five patients who died of hypoglycemia.

At the meeting of the Section of Neurology of the American Medical Association, June 7, Moersch and Kernohan, of Rochester, Minnesota, read an authoritative paper entitled "Hypoglycemia: Neurologic and Neuropathologic Study." They describe carefully the clinical picture ot a patient going into insulin shock. First there is a short period of lassitude with mental retardation, mild confusion and sometimes diplopia. Then, with increasing pallor and profuse perspiration, the patient may become excited and delirious, finally going into coma. This may last only a few minutes or be prolonged for hours. It is usually at this stage that convulsive phenomena appear with many reflex changes and even transient motor paralysis. Dextrose is then given and "the rapidity of recovery is frequently incredible. These patients may appear to be snatched from the very doors of death." Recovery usually appears to be complete, but occasionally there is residual paralysis, aphasia and mental dullness lasting a few days.

The latter authors have carefully observed the effect of hypoglycemia in 44 patients who had more than 300 severe hypoglycemic reactions. Two of the patients died. The brain of the first showed edema and widespread degeneration of nerve cells in the cerebral cortex and basal ganglia, with acute changes in the microglia and oligodendroglia. "There were practically no normal nerve cells in the cerebral cortex." In certain devastated areas no nerve cells remained, and there was degeneration of the underlying white matter. In the second case the histologic examination was less complete, but again there was a conspicuous loss of cells in the cerebral cortex and hind-brain, the tissue looked edematous, and extravasation of red blood cells into perivascular spaces was found.

These two cases had suffered from many and severe hypoglycemic reactions due to hyperinsulinism, so the insult to the brain was probably greater than that from the induced hyperinsulinism used for therapy. Some of the changes described in the nerve cells are doubtless reversible, but it seems possible that insulin shock destroys a great number of cerebral nerve cells by some mechanism akin to asphyxiation. Experimental work on animals [4,5] has shown that insulin convulsions may cause hemorrhages into the brain. The basal ganglia are the chief seat of the hemorrhagic lesions, although they often occur in the cerebral cortex.

Taking these observations into account, and critically examining the reports of "cures" of schizophrenia by insulin shock, one is obliged to be skeptical. One suspects that the treatment is merely palliative and carried out at the risk of permanent damage to the brain.

STANLEY COBB, M.D.
Massachusetts General Hospital,
Boston, Mass.

REFERENCES

1. Cotton, H. A.; Draper, J. W., and Lynch, J. M.: Intestinal pathology in the functional psychoses, preliminary report of surgical findings, procedures and results. Med. Rec. 97:719 (May 1) 1920.
2. von Meduna, L.: Versuche über die biologische Beeinflussung des Ablaufes der Schizophrenie; Campher- und Cardiazolkrämpfe. Ztschr. f. d. ges. Neurol. u. Psychiat. 152:235, 1935.
3. d'Elseaux, F. C., and Solomon, H. C.: Use of carbon dioxide mixtures in stupors occurring in psychoses. Arch. Neurol. & Psychiat. 29:213 (Feb.) 1933.
4. Baker, A. B., and Lufkin, N. H.: Cerebral lesions in hypoglycemia. Arch. Path. 23:190 (Feb.) 1937.
5. Stief, A., and Tokay, L.: Beitrage zur Histopathologie der experimentellen Insulinvergiftung. Ztschr. f. d. ges. Neurol. u. Psychiat. 139:434, 1932.

THE COMMONWEALTH OF MASSACHUSETTS

BOARD OF REGISTRATION IN MEDICINE

To the Editor: At the annual meeting of the Board of Registration in Medicine held in July, Dr. Francis R. Mahony, of Lowell, was re-elected chairman and Dr. Stephen Rushmore, of Boston, was re-elected secretary.

STEPHEN RUSHMORE, M.D., *Secretary.*
State House, Boston.

REPORT OF MEETING

SPECIAL LECTURES IN ENDOCRINOLOGY

The "Physiology of Menstruation" was the subject of one of the Special Lectures in Endocrinology delivered at the Harvard Medical School by Dr. F. L. Hisaw on March 11, 1937. Dr. Hisaw discussed the use of hormones to regulate the development of the female reproductive tract of the *Macacus rhesus* monkey, basing his remarks upon two groups of experiments: (1) those dealing with the action of estrin and progesterone after castration, and (2) those in which modifications of the reproductive tract were induced through the action of anterior pituitary preparations on the ovaries. It is possible by both of these methods to produce conditions typical of the different phases of the menstrual cycle.

Uterus. Conditions characteristic of the follicular half of the cycle can be restored after castration atrophy and maintained for an indefinite time by the injection of estrin. However, if the maintenance dosage of estrin is 100 r.u. (rat units—Allen and Doisy) or less, daily uterine bleeding may occur by the thirtieth or fiftieth day, but if 200 r.u. or more are given, bleeding usually does not take place during the treatment. If a sufficient amount of estrin to restore the normal condition of the endometrium is given, bleeding almost invariably follows discontinuance of the injections. Bleeding can also be precipitated by lowering the dosage of estrin. In all instances when bleeding occurs during or following injections of estrin, it results from disintegration of an endometrium which is morphologically typical of the follicular half of the menstrual cycle.

All stages in the development of a premenstrual endo-

metrium, similar in all respects to that found during the corpus luteum half of the cycle, can be induced in castrated monkeys by injecting the corpus luteum hormone, progesterone. When progesterone is given following a series of injections of estrin, the mucous membrane of the glands and surface epithelium swells, forms glycogen which is discharged from the cells and becomes secretorially exhausted within about 20 to 25 days, and remains in this condition as long as the treatment is continued. Injections of progesterone will also produce a fully developed premenstrual endometrium after atrophy following castration and without a previous treatment with estrin, provided a sufficiently large dosage of the corpus luteum hormone is injected. A smaller dosage of progesterone is required to produce the corpus luteum effects if it is preceded by or given simultaneously with estrin. Furthermore, a better development of the endometrium can be induced by giving estrin and progesterone simultaneously than by injecting either hormone alone.

Inhibition of Menstruation. The uterine bleeding which usually follows castration of adult monkeys can be inhibited by estrin provided the ovaries are removed during the follicular half or early lutein phase of the cycle. When the animals are castrated during the luteal half of the cycle, postoperative bleeding becomes progressively more difficult to inhibit by the injection of estrin, as the endometrium is transformed into a premenstrual condition. In contrast with this, progesterone will inhibit postoperative bleeding following castration at any time of the cycle.

The ability of estrin and progesterone to inhibit uterine bleeding can be tested in another way. When 100 r.u. of estrin are given daily to castrated monkeys for 20 days they will bleed within a few days if the treatment is discontinued. If the dosage is reduced to 25 r.u. daily, bleeding occurs on about the sixth day, and 50 r.u. daily will postpone bleeding until about the ninth or tenth day. When progesterone is given following a 20-day treatment with estrin, as described above, bleeding usually begins on the sixth day with ¼ rb.u. (rabbit unit—Allen and Corner) daily, on the eighth to thirteenth day with ½ rb.u., and on the fifteenth to twenty-fifth day with ¾ rb.u.; 1 rb.u. daily has been given for 44 days without bleeding, but menstruation began 4 to 7 days after the dosage was reduced to ½ rb.u. daily.

It has also been found that after a prolonged treatment with estrin, dosages of estrin and progesterone which when given alone will not inhibit bleeding will do so when the two hormones are injected simultaneously. When 25 r.u. plus ¼ rb.u. are given daily, bleeding begins between the eighth and fourteenth days; 25 r.u. plus ½ rb.u. may be continued 18 to 22 days without bleeding. With 50 r.u. plus ¼ rb.u. bleeding may not occur within 28 days, and 50 r.u. plus ½ rb.u. may be given for 20 to 22 days without bleeding. It was also found that ¼ rb.u. of progesterone plus 25 to 50 r.u. of estrin injected daily for 21 days will not produce a fully developed premenstrual endometrium, while ½ rb.u. plus the same dosage of estrin and given for the same length of time will do so.

Implantation Sites. Endometrial proliferations that seem to be identical with normal implantation sites of fertilized ovums can be induced by traumatizing the endometrium while it is under the influence of the corpus luteum hormone. The proliferations that are initiated at the points of injury usually spread, and by the eighth day involve the entire inner portion of the endometrium bordering the uterine lumen. Such cells originate from the surface epithelium of the uterine lumen and from the epithelium of the glands, and grow out into the surrounding stroma in clumps. Many cells reach the proportions of giant cells and fuse with their neighbors, producing multinucleated masses.

The proliferations seem to attain their maximum development during the second week following traumatization, and by the fifteenth day show marked degenerative changes, especially in regions adjoining the uterine lumen.

Glycogen Deposition. The appearance of glycogen in the epithelial cells of the surface mucosa and glands of the endometrium is usually associated with the luteal half of the cycle. Glycogen formation and secretion can be elicited by the injection of progesterone, and even when the treatment is continued for 30 days or longer and the endometrium reaches a condition of secretory exhaustion, some glycogen is always found. Estrin in moderate doses (80 r.u. or less daily) does not usually stimulate glycogen deposition unless the treatment is continued for a long time; large doses (200 to 500 r.u. daily), however, produce glycogen storage in the epithelial cells, but little or none of it seems to be released into the lumen of the glands, at least in a stainable form. If relatively small amounts of progesterone (½ rb.u. or more daily) are injected simultaneously with estrin, both glycogen formation and secretion occur. From these observations it seems that progesterone can bring about both formation and secretion of glycogen, while estrin, if given in sufficient dosage, causes chiefly glycogen storage.

Cervix. Overholser and Allen (1933) have called attention to a condition of squamous metaplasia in the cervical glands of monkeys which follows prolonged estrin administration; this observation has been repeatedly confirmed by others. When progesterone is given simultaneously with estrin the formation of these metaplastic bodies are inhibited; if already present, as a result of previous injections of estrin, they disappear when progesterone is added to the treatment. In addition, these cervical lesions apparently undergo regression spontaneously when all treatment is discontinued.

Progesterone also seems to inhibit the growth-promoting action of estrin on the cervix. When the two hormones are given together the cervix does not attain the size usually found when estrin is given alone.

Vagina. Davis and Hartman (1935) have shown that the vaginal epithelium of monkeys becomes greatly thickened and cornified during the follicular half of the cycle, while after ovulation, desquamation begins and increases in rate as menstruation approaches. They also found that similar though more pronounced changes occur in the vaginal epithelium during pregnancy. After the middle of pregnancy the functionalis is gradually lost, and during the last few weeks prior to parturition all that remains of the mucosa is a few layers of basalis of irregular thickness.

These changes can be produced in a castrated monkey by injecting estrin and progesterone. It is well known that estrin alone will produce growth and cornification of the vaginal mucosa. Progesterone, in contrast to estrin, has little or no effect on the vaginal mucosa, but it does modify the action of estrin when both hormones are given simultaneously. When progesterone is added to the treatment, after estrin has been given alone for several days, the cornified layers of the vaginal mucosa are gradually lost and the epithelium becomes greatly reduced in thickness. If progesterone is given first for a few days and then estrin is added to the treatment, the vaginal mucosa remains in a condition resembling that of a castrated animal. Such experiments show that progesterone can modify and inhibit the action of estrin on the vaginal epithelium.

Sexual Skin. The marked edema of the sexual skin, a

characteristic effect of estrin in juvenile monkeys, can be inhibited by simultaneous injections of progesterone.

Effects of Pregnancy Urine Hormone on the Action of Estrin and Progesterone. When pregnancy urine hormone is given simultaneously with estrin, the effects on the uterus and vagina are the same as those found when estrin is injected alone. The cervix, however, is much larger when the two hormones are combined. As has been mentioned, progesterone decreases the action of estrin on the cervix, so that when it is added to the treatment the synergism between estrin and the pregnancy urine hormone is inhibited, and as a result the cervix is smaller than when estrin is given alone.

Under the discussion of the action of progesterone and combinations of progesterone and estrin on the uterus it was stated that the epithelium of the glands passes through a secretory phase, as shown by fraying of the cells, and returns to a relatively inactive condition when the treatment is continued for longer than about 20 days. The addition of pregnancy urine hormone to the treatment prolongs the secretory phase both when progesterone is given alone and when progesterone is combined with estrin.

These observations indicate that pregnancy urine hormone increases the action of estrin on the cervix and augments the effects of progesterone on the endometrium.

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Monday, August 2

Wednesday, August 4—

†12 m. Clinicopathological conference. Children's Hospital Amphitheater.

Friday, August 6—

12 m. Massachusetts General Hospital. Clinical meeting of the staff of the Children's Medical Service. Ether Dome.

Saturday, August 7—

•10 a. m.-12 m. Staff rounds at the Peter Bent Brigham Hospital. Conducted by Marshall N. Fulton.

•Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

September 13-17—Fifth International Congress of Radiology. Page 709, issue of April 22.

September 21, 22 and 23—Thirteenth Clinical Congress of the Connecticut State Medical Society, New Haven.

October 1 and 2—New England Surgical Society. Providence, R. I.

October 5-8—American Public Health Association meeting. New York City.

October 9—American Board of Ophthalmology, Chicago. Page 722, issue of April 22.

October 19, 20, 21—Academy of Physical Medicine. Page 723, issue of April 22.

October 25-29—American College of Surgeons. Chicago, Illinois.

November 1-12—1937 Graduate Fortnight of the New York Academy of Medicine. Page 1000, issue of June 3.

November 6—American Board of Obstetrics and Gynecology. Page 153, issue of July 22.

April 4-8, 1938—The American College of Physicians. Page 41, issue of July 1.

June 13 and 14, 1938—American Board of Obstetrics and Gynecology. Page 153, issue of July 22.

BOOKS RECEIVED FOR REVIEW

Emanotherapy. F. Howard Humphris and Leonard Williams. 188 pp. Baltimore: William Wood & Company, 1937. $3.00.

The Treatment of Diabetes Mellitus. Elliott P. Joslin, with the cooperation of Howard F. Root, Priscilla White, and Alexander Marble. Sixth Edition, Thoroughly Revised. 707 pp. Philadelphia: Lea & Febiger, 1937. $7.00.

The Normal Encephalogram. Leo M. Davidoff and Cornelius G. Dyke. 224 pp. Philadelphia: Lea & Febiger, 1937. $5.50.

Infantile Paralysis and Cerebral Diplegia. Methods used for the restoration of function. Elizabeth Kenny. 125 pp. Sydney, Australia: Angus & Robertson, Limited, 1937. £1 1s.

International Clinics. Edited by Louis Hamman. Volume II. Forty-Seventh Series. 315 pp. Philadelphia, Montreal, London: J. B. Lippincott Company, 1937. $3.00.

The Larynx and Its Diseases. Chevalier Jackson and Chevalier L. Jackson. 555 pp. Philadelphia and London: W. B. Saunders Company, 1937. $8.00.

Short-Wave Diathermy. Tibor de Cholnoky. 310 pp. New York: Columbia University Press, 1937. $4.00.

Personal Hygiene. C. E. Turner. 335 pp. St. Louis: The C. V. Mosby Company, 1937. $2.25.

Fifty-Ninth Annual Report of the Department of Health of the State of New Jersey, 1936. 431 pp. Trenton: MacCrellish & Quigley Company, 1936.

A Textbook of Applied Biochemistry for Pharmacists and Pharmaceutical Students. Frank Wokes. 522 pp. Baltimore: William Wood & Company, 1937. $5.00.

The Endocrines in Obstetrics and Gynecology. Raphael Kurzrok. 488 pp. Baltimore: The Williams & Wilkins Company, 1937. $7.50.

BOOK REVIEW

The Larynx and Its Diseases. Chevalier Jackson and Chevalier L. Jackson. 555 pp. Philadelphia and London: W. B. Saunders Company, 1937. $8.00.

Heretofore the outstanding book on the diseases of the larynx has been that of Sir Sinclair Thomson. The surpassing of even this classic would seem to have been accomplished by the Chevalier Jacksons whose "Larynx and Its Diseases" brings this subject up-to-date with a completeness of detail which by present standards leaves little room for improvement. The vast and unrivaled experience of the author and his filial collaborator has enabled them to cover in a text of 500 pages, with 221 illustrations (11 of them color plates), every form of laryngeal pathology. The arrangement is in the conventional style with an initial chapter on anatomy and physiology, another on symptomatology, diagnosis and general consideration of treatment. There follow chapters on anomalies and malformations, noninflammatory edema of the larynx, wounds, dislocations, fractures, and acute and chronic inflammations. To syphilis and tuberculosis of the larynx are devoted separate chapters. Laryngeal pathology due to fungus infections and that associated with dermatoses, drug ingestion and general diseases are covered in unusual detail. Three chapters comprise the discussions of laryngeal neuroses and paralyses. Benign and malignant tumors are fully considered, and the final four chapters are given over to the exposition of endolaryngeal and external operations with particular emphasis on the procedures for cure of malignant diseases of the larynx.

Almost classical is the discussion of emergency and orderly tracheotomy, and to the former is added the rare touch of a "candid" view of the senior author. As usual the colored plates are from the personal brush of the authors, thus assuring accurate reproductions of actual clinical observations. The legends are particularly valuable in summarizing the important data with regard to the cases illustrated.

The book will find its greatest usefulness as a reference book for practitioners of laryngology. For the general practitioner it would seem perhaps to be bewilderingly complete and hence to some extent to fail to reach a group for which it was, among others, specifically intended.